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A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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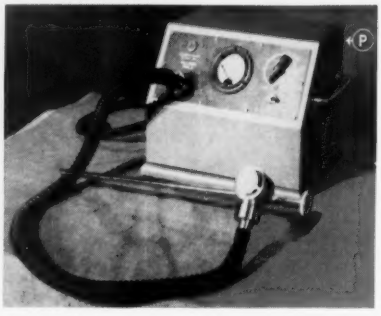

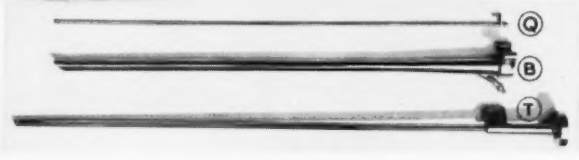

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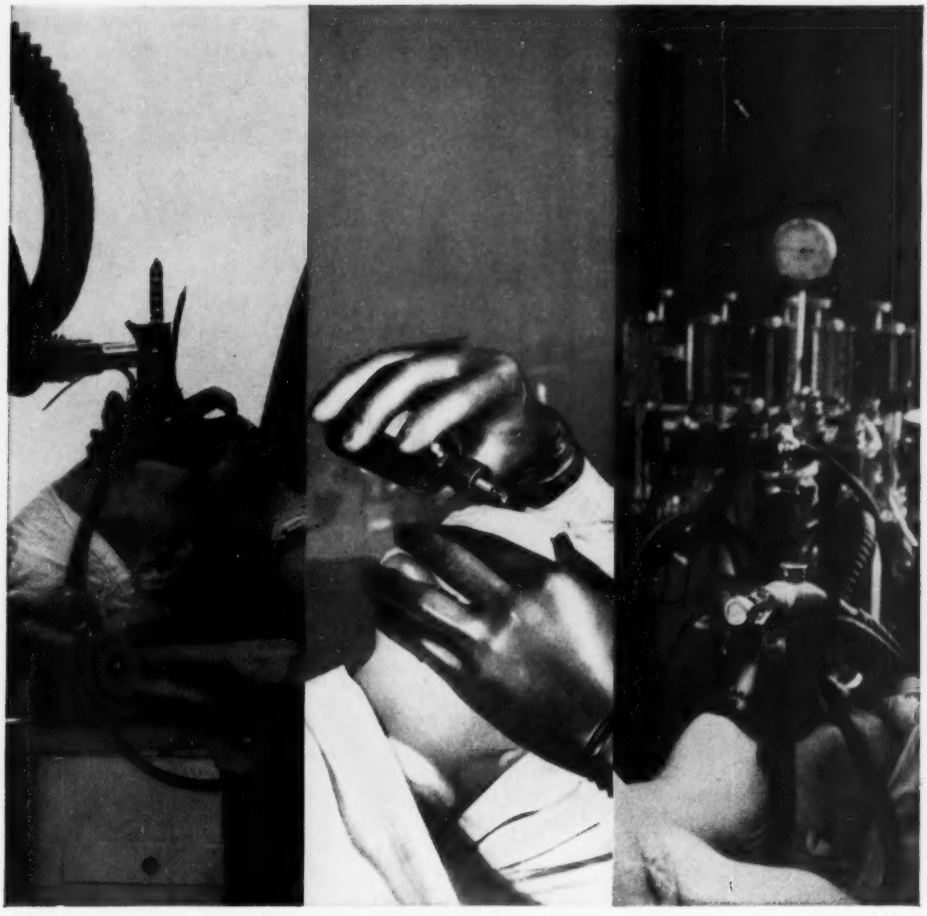

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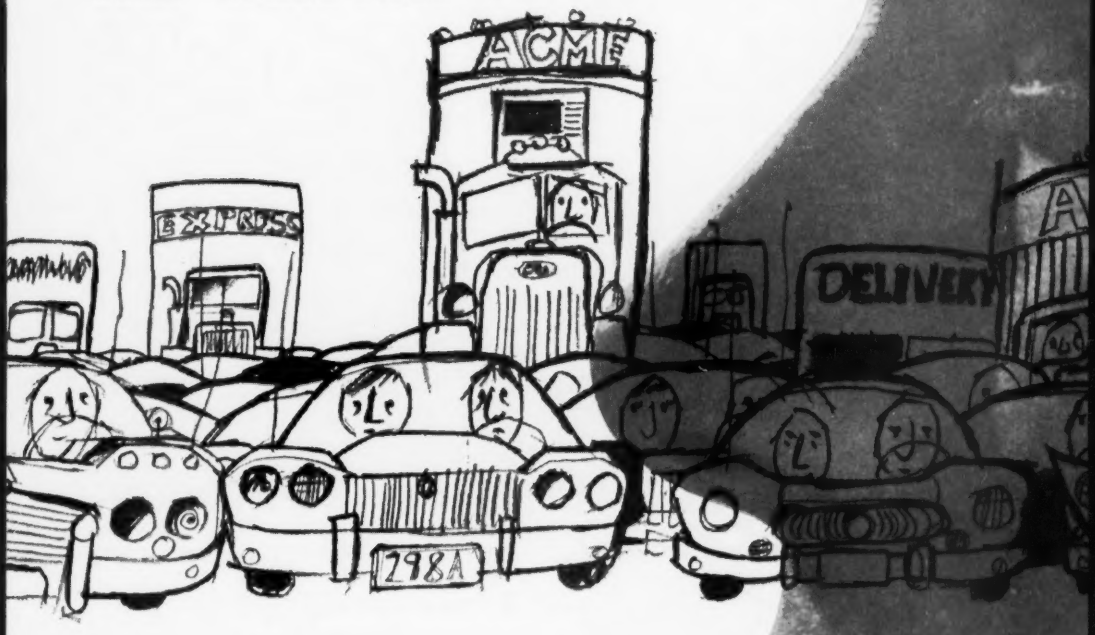

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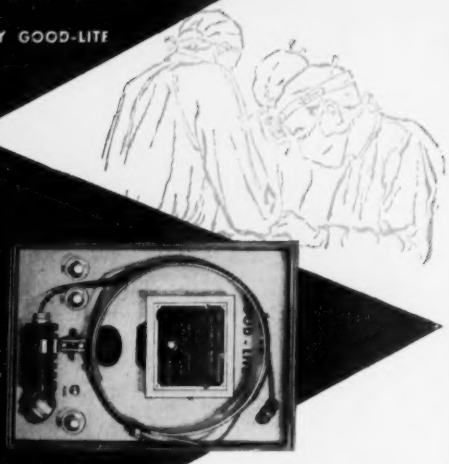

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A.M.A. ARCHIVES OF
OTOLARYNGOLOGY

Vertigo

M. M. HIPSKIND, M.D., Chicago

Everything on earth is in motion with reference to any fixed point in space. Motion, therefore, is a primary force in life; indeed, absence of motion is death. Man moves and is affected by motion.

The master mechanism controlling our motion is a modest little special sense organ housed in the walls of the labyrinth deep within the protective ramparts of the temporal bone in the middle of the skull. The fact that the labyrinth is the only organ in the body which has reached its full size and development at the time of birth is significant. The skull of early man shows the labyrinth has not been modified by nature during the thousands of years of man's effort to conquer the physical world. Man alone has the power of speech. Full exploitation of speech is dependent upon hearing. Again it is significant that the labyrinth is the seat of the receptor for hearing.

We recognize the heart as a remarkable mechanism. Its efficiency is evidenced by the fact that it pumps blood throughout the circulatory system from birth until death. The labyrinth also begins to function at birth, and continues in a constant state of tone and action throughout life, awake or asleep, day and night. However, it does not share the heart's privilege of resting 60 to 80 times each minute.

Submitted for publication Aug. 8, 1958.

Read before the Section on Laryngology, Otology, and Rhinology at the 107th Annual Meeting of the American Medical Association, San Francisco, June 27, 1958.

All this would suggest that the labyrinth is the seat of an "extra-special" sort of sense organ. As far as our position in space is concerned, all is well until something goes wrong with the vestibular labyrinth or its central connections. Cawthorne [1] tells us, "The overwhelming giddiness, the awful sickness, and the turbulent eye movement—all enhanced by the slightest movement of the head—that accompany acute vestibular failure form a picture of helpless misery that has few parallels in the whole field of injury or disease."

For our purpose we define vertigo as a hallucination of movement, in other words, the patient sees or feels movement that is not there. What are the causes of vertigo? From Cawthorne's series of 1,523 cases, the relative frequency according to location of the lesion is as follows:

Central		107
Peripheral		1,349
Hydrops (Ménière's disease)	972	
Trauma	123	
Vestibular neuronitis	109	
Positional	84	
Infective	61	
Not classified		67
Total		1,523

The patient's greatest fear is that his vertigo is due to a serious brain disease or forecasts a stroke. From the above statistics it is evident that there is a ten-to-one chance that his trouble is in the peripheral organ. If the physician can rule out brain tumor as a basis for the vertigo, he may

reassure the patient. The "peace of mind" experienced by the patient who is assured that his dizziness is not due to serious brain disease is more beneficial in the management of vertigo than all the sedatives and tranquilizers the physician may prescribe.

An acute vestibular episode may cause a flow of abnormal impulses spreading from the vestibular nuclei in the brain stem to the adjacent vagal centers, producing pallor, nausea, vomiting, headache, slow thready pulse, and a fall in blood pressure. All of these symptoms are commonly misinterpreted as evidence of brain disease leading to unnecessary spinal puncture, pneumoencephalography, and other procedures.

These patients want to tell their story in detail. In order to gain the confidence of the patient, the physician must be willing to take an adequate history and make an orderly and complete examination. Temperature, pulse, respirations, and blood pressure should be recorded. The ears, nose, and throat must be carefully examined. Complete examination requires audiometry, pure-tone and speech tests, caloric and turning tests, examination of the cranial nerves, and tests for Rhomberg, gait, motor strength, reflexes, adiadochokinesis, and past-pointing. When indicated, x-ray studies should be made of the suspected areas, such as the mastoid, petrous pyramids, internal acoustic meatuses, and sinuses. If a central lesion is suspected, special neurological phases should be carefully evaluated. In unclassified cases of vertigo, one is well advised to seek aid from consultants in special fields. The otologist, neurologist, internist, and ophthalmologist are important members of the team concerned with the problem of vertigo.

One of the most helpful examinations in the study of the nature of the vertigo is the test for positional nystagmus.

The postural tests are carried out according to the method of Cawthorne.[2] The patient is seated upon the examination table. He is told he will be placed in various positions, and that it is imperative that he keep the eyes open at all times. Frenzel glasses are used, and the patient is instructed to look directly forward. Spontaneous nystagmus is noted, if present. If absent, the patient's head is held by the examiner, and the patient is placed in the supine position with the head to one side (the side indicated as the offending position if given in the history). A latency period of 10 seconds is permitted for the recognition of positional nystagmus. If nystagmus does not occur after 10 seconds, the patient is brought upright, and again the examiner looks for nystagmus. If absent after 10 seconds, the patient is placed in the supine position to the opposite side of the first procedure. If nystagmus is not observed, the patient is returned to the sitting position and again examined for the presence or absence of nystagmus. In the absence of nystagmus, the patient is placed in the supine position with the head lower than the shoulders (midline, head-hanging position). If nystagmus is absent after 10 seconds, the patient is returned to the upright position and again examined for nystagmus.

If nystagmus occurs, the intensity, direction or nature, the duration and the presence or absence of vomiting are noted on the record. If nystagmus is noted in one or more of the positions, that position is held until it subsides. If the nystagmus persists for more than 30 seconds, it will probably continue as long as the critical position is maintained. Should nystagmus persist for 30 seconds, the patient is returned to the upright position, and after a short pause is placed in the same position which provoked the nystagmus. If the nystagmus does not reappear, it is recorded as *readily fatigable*. If the nystagmus reappears with diminished intensity, and on further attempts does not occur at all, it is recorded as *gradually fatigable*, and if the nystagmus continues with undiminished intensity despite repetition, it is recorded as *nonfatigable*.

Characteristics of Cases Belonging to the Peripheral and Central Groups *

	Peripheral Group	Central Group
Direction of nystagmus	Constant in different head positions	May vary with different head positions
Onset of nystagmus after assuming new posture	Latency period of 3-10 seconds	Immediate onset
Amplitude of nystagmus	Usually large	Usually small
Duration of nystagmus	3-10 seconds	Much longer, may continue indefinitely as long as provocative posture is maintained
Fatigability of response to repetition of postural change	Nystagmus cannot be elicited more than 1, 2, or 3 times in diminishing intensity	Nystagmus recurs each time provocative posture is assumed
Sensation of vertigo	Severe	Mild
Associated nausea	Common	Rare

* From H. F. Schuknecht.

Additional differentiation may be classified as shown in the Table.

Dizziness is one of the earliest symptoms the patient with multiple sclerosis may experience. A sclerotic plaque may be deposited in the area of the eighth nerve. Intention tremor, scanning speech, optic disk changes, and a paretic gold curve in the presence of a negative Kahn reaction suggests the diagnosis.

Dizziness described as a feeling of rotation or bodily displacement is common in the epileptic. Aura, loss of consciousness with cry, fall, and tonic or clonic convulsions make the diagnosis.

Acoustic neuroma is frequently ushered in by slight dizziness. The diagnosis is indicated by the gradual onset of a unilateral hearing loss progressing to total deafness. The patient falls (Rhomberg) to the side of the lesion, and nystagmus is toward the affected side. The caloric test in the late stage is absent on the affected side. The enlargement of the internal acoustic meatus shown on x-ray makes a positive diagnosis.

The patient with supratentorial brain tumor usually has a persistent sense of disequilibrium. He has a postural Type 1 nystagmus (rarely Type 2), and, if the provocative position is maintained, he may vomit during the course of the postural tests. Failing vision, choking of the disks of the optic nerve, with sensory changes, point to the diagnosis. Nystagmus of a vertical nature strongly suggests a brain tumor. Infratentorial (cerebellum-brain stem) tumors are characterized by a disturbance of equilibrium and coordination. The Rhomberg is positive; past-pointing and adiadochokinesis are present. The nystagmus may be spontaneous, is usually fine and continuous. If the nystagmus is postural, it will recur each time the provocative position is assumed and will continue as long as the patient remains in the offending position. The gait is ataxic.

Interference with the blood supply to the cerebrum, cerebellum, and brain makes up the largest group of patients presenting dizziness as a chief complaint. Vertigo may be a symptom of hemorrhage, thrombosis, embolism, vasomotor insufficiency, hypertension, hypotension, the climacteric, the side-effects of drugs—specifically opiate derivatives, streptomycin, quinine, salicylates, barbiturates—and postanesthesia. It may also be a symptom of intoxications from alcohol, tobacco, lead, DDT and lindane. Postural vertigo and incapacitating dizziness may be a prominent feature of postcerebellar artery thrombosis, meningovascular syphilis, arteriosclerosis, periarteritis nodosa, thromboangiitis nodosa, thromboangiitis obliterans, thrombocytopenic purpura, leukemia, pernicious anemia, and polycythemia vera. Each of the disease entities may be identified by proper tests. The important point is awareness of the clinician that dizziness may be a part of these diseases.

Virus infections, particularly measles, mumps, scarlet fever, typhoid, and typhus, and Ramsay-Hunt disease may be the basis for dizziness. In encephalitis the dizziness is associated with an unsteady gait, diplopia, dysphagia, drooling of saliva, occasionally facial paralysis, and frequently stupor.

All the magic of antiquity fails to match the marvels of today's chemical laboratory. In the last few decades the span of life has been increased by 15 years. Infants and the aged survive through the application of modern chemicals and techniques, transfusions, antibiotics, and chemotherapeutic agents. Surgery replaces worn-out and diseased tissues by transplants and shunting procedures. Nutrition approaches an exactness of a mathematical science. All of this, associated with an acceleration of our lives by modern techniques in education, industry, agriculture, and transportation, has placed us in a vulnerable position. We become tense, nervous, and emotional. Dizziness is an outstanding feature of the anxiety state. In the absence of localizing signs of peripheral or central disease, a functional basis must be postulated for the vertigo and appropriate treatment instituted.

In 90% of patients complaining of giddiness, light-headedness, post-in-a-hole, whirling, spinning, ship-board sensations, and merry-go-round feelings, the trouble will be found in the labyrinth and/or its central connections. The commonest lesion is labyrinthine hydrops (Ménière's disease). Tinnitus and unilateral hearing loss are the two outstanding signs for localization of the lesion. In 10% of the cases the disease may be bilateral. Recruitment is the rule.

The vertigo is postural in character; the onset sudden and of short duration. The nystagmus is Type 2; in other words, the direction of the nystagmus does not change with different head positions. The amplitude of the nystagmus is large, and it is of the "readily fatigable" type. Caloric tests show a reduced reaction on the affected side. The attacks are marked by periodicity; the patient has freedom of dizziness between attacks and experiences relief after rest.

Dizziness after head injury is a common complaint. It is agreed that nystagmus cannot be voluntarily produced. It is further agreed that vestibular nystagmus is always associated with vertigo. Therefore, if postural tests provoke nystagmus, the clinician has objective evidence of the patient's subjective complaint of dizziness. If the patient is observed in the period shortly after the injury, blood may be observed in the middle ear in cases of longitudinal or transverse fracture. Occasionally spinal fluid may be observed. The hearing loss due to blood in the middle ear will return to the preinjury level after the absorption of the blood, usually within three weeks. Tinnitus may subside in three weeks, or may persist.

In labyrinthine concussion a high-tone loss similar to the 4,096 dip observed in acoustic trauma may be demonstrated. In a round table discussion during the Triologic Meeting in Chicago, Schuknecht expressed the opinion, "If positional nystagmus is demonstrated after head injury, a 4,096 dip will be present."

Otitis media, catarrhal or suppurative, may provoke dizziness. The suppurative course may progress to fistula of the labyrinth. A positive fistula test demands surgical intervention if the spread of the infection to the intracranial area is to be prevented.

Vestibular neuronitis is a term used by Dix and Hallpike[3] to describe what they consider to be a septic process affecting the vestibular apparatus, the peripheral nervous pathways up to and including the vestibular nuclei in the brain stem. The dizziness is postural and similar to labyrinthine hydrops except for the absence of tinnitus and deafness. The course of the vertigo is usually benign, and it subsides in a few weeks. The prominent feature is the evidence of an infection as indicated by an elevation in the white blood count and sedimentation rate. A septic process usually is found in the nose, sinuses, or throat. A reduced response to galvanic stimulation is considered to be diagnostic.

In my experience, paroxysmal positional vertigo is one of the most frequent types of dizziness. Bárány described this dramatic form of episodic vertigo in 1921. He believed the pathology was located in the

otolithic organ in one type, and placed the lesion in the central nervous system in a second type. Wherever the lesion, these cases are marked by a violent postural vertigo. The nystagmus comes on after a latency period of only a few seconds; the amplitude is large, and the subjective vertigo so severe that the patient will frequently cry out and grasp for support. The episode passes quickly and usually cannot be reproduced except after a period of at least 30 minutes rest. The main feature is the "fatigability" of the postural vertigo in a case free of tinnitus and deafness.

Dizziness may be associated with many other conditions, e. g., foreign bodies and cerumen in the external canal, and barometric changes affecting the middle ear pressure. Transient dizziness may be associated with auricular fibrillation, decompensated cardiac disease, aortic stenosis, and carotid sinus sensitivity.

Vertigo is a frequent and troublesome experience to the patient. His attacks make gainful employment impossible and fill his conscious hours with fear, and not infrequently are so severe that they awaken him from sleep. In the care of these patients the physician has an unparalleled opportunity to treat the sick and to aid the halt. Through proper diagnosis and treatment many of these patients may be returned to a full, useful life.

The expression used in a meaningless way before the age of earth satellites "out of this world" has taken on new and sober tones. We must familiarize ourselves with the mechanism governing our equilibratory activities. Cooley [4] asks, "Will motion sickness plague space travelers when there is no up and down?" How does the body react to the eerie feeling of weightlessness which space travelers will encounter? All these problems are locked in the function of the vestibular apparatus and its central connections. If man is to triumph in his conquest of space, a greater knowledge of the tiny labyrinth must be weighed equally in importance with the weight of the four-story-high intercontinental missile.

55 E. Washington St. (2).

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Neomycin Ototoxicity

Report of a Case

GLENN J. GREENWOOD, M.D., Los Angeles

Since streptomycin, with its salts, has demonstrated such tuberculostatic potential, many authors have cited its ototoxic propensity, viz.: Fowler and Seligman [1]; Carr et al.[2]; Shane and Laurie [3]; Glorig [4]; Heck, Lynch, and Graves,[5] etc. Now another "mycin," neomycin, primarily bactericidal, adds to this ototoxic incidence. Preservation of human life commands continued "mycin" use, even at the risk of hearing impairment. Detoxifying methods, will, I trust, in the future, permit continued use of the systemic therapeutic potential of neomycin.

Waisbren avers that neomycin has the widest antibacterial spectrum of any of the antibiotics. It is primarily bactericidal. It is effective against many Gram-negative and Gram-positive bacteria and virtually all strains of Micrococcus pyogenes. It is least effective against Proteus, Pseudomonas aeruginosa, Pseudomonas species, and enterococci. However, it remains the best drug available against the Pseudomonas species.[6]

As bacterial resistance to other antibiotics and chemotherapeutic agents mounts, the use of neomycin topically for superficial skin (and eye) infections and orally for preoperative bowel preparation, steadily increases. Only small amounts are required topically. Less than 3% is absorbed from the intact, unobstructed intestinal lumen.[7] It has a low sensitizing potential, and infrequently produces bacterial resistance.[6] Apparently increasing too, though unestablished, is its current use for local application in the urinary tract [8] and as a cold sterilizing agent for urological instruments.[9] As an aerosol, Waisbren believes it to be the most effective antimicrobial agent in the management of patients with chronic bronchitis, bronchiectasis, and emphysema.[6]

Neomycin is so toxic that its parenteral use is contraindicated except in certain cases. When so used it should be by intramuscular route only. Respiratory depression has been reported when it was instilled intraperitoneally in the presence of ether and is due to the blocking action of neomycin at the neuromuscular junction. Pittinger and Long recently have shown that experimentally, neostigmine methylsulphate effectively antagonizes this neuromuscular blocking mechanism and respiratory depression.[10]

Neomycin exerts a most toxic action upon the collecting tubules of the kidneys [11]; and the hair cells of the cochlea, the vestibular hair cells being implicated to a much slighter extent, as shown experimentally in animals.[12] Where the intramuscular route is commandeered, these critically ill patients require hospitalization for lysing pathogens resistant to other antibiotics. A not insignificant number of these patients are encountered after reconstructive surgery of the genitourinary and/or intestinal tracts.

Efforts have been made in experimental animals to lessen the ototoxicity of neomycin sulfate by substituting pantothenic acid as the calcium pantothenate.[13] While some of the acute toxicity may be lessened by the addition of ionized calcium to pantothenic acid, the over-all chronic toxicity

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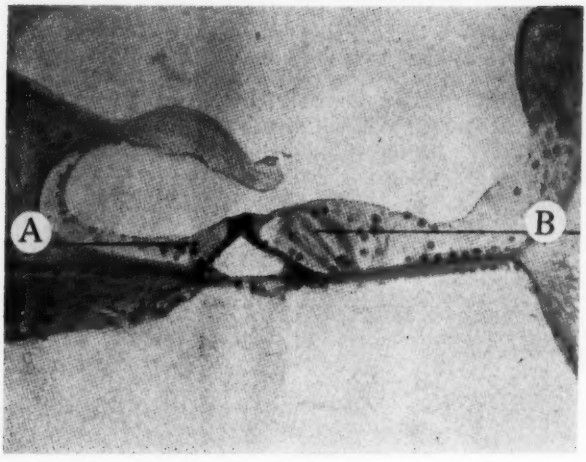


Fig. 1 (Animal 1408).—Organ of Corti, first turn cochlea. Neomycin 100 mg/kg/day s. c., 30 doses. Degeneration of internal (*A*) and external (*B*) hair cells. Second and third turns also have complete degeneration of internal and external hair cells; ×300. (Reproduced with permission from Hawkins, J. E., and Lurie, M. H.: Ann. Otol. Rhin. & Laryng. 62: 1128, 1953.)

that pantothenic acid accords ectoderm is not.[14-17] These investigators, however, in their experiments, did not use neomycin pantothenate but the related "mycins," streptomycin and dihydrostreptomycin pantothenate.

The ototoxicity of neomycin, as shown by Hawkins and Lurie in electrophysical studies of hearing in the cat, apparently initially degenerates the external and later the internal hair cells of the organ of Corti, but degeneration of both may be complete. However, complete dissolution of the organ of Corti was not seen. This degeneration is greatest in the basal coil but may be scattered throughout the cochlea. The vestibular system of a neomycin-treated cat (No. 1365) revealed only a slight scattered hair cell loss of the crista, when viewed under high-powered magnification.[12] Smaller doses of neomycin were here used.

Report of Case

On Sept. 25, 1957, a 38-year-old Caucasian housewife was admitted to the U. C. L. A. Medical Center on the Urological Service. She gave a history of being hospitalized in November, 1948, while residing in the east, for radium therapy of a carcinoma of the cervix. Some four years and two months after this therapy, a reoccurrence of her symptoms necessitated a radical hysterectomy and a pelvic lymphadenectomy. During her postoperative course, a ureterovaginal fistula and a bilateral hydronephrosis developed. There then

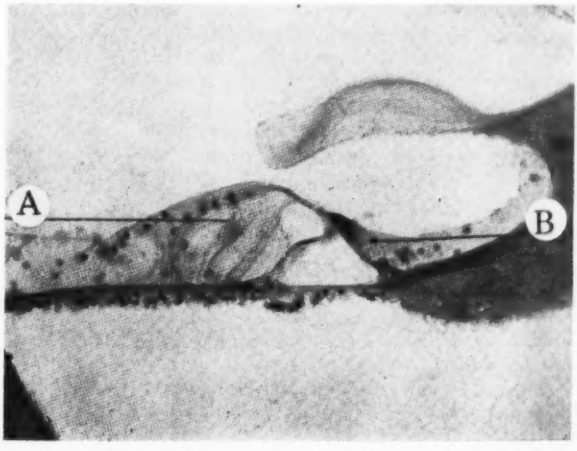


Fig. 2 (Animal 1448).—Organ of Corti, first turn cochlea. Neomycin 80 mg/kg/day s. c., 15 doses. Complete degeneration of internal (*A*) and external (*B*) hair cells. Second and third turns also have complete degeneration of internal and external hair cells; ×300. (Reproduced with permission from Hawkins, J. E., and Lurie, M. H.: Ann. Otol. Rhin. & Laryng. 62: 1128, 1953.)

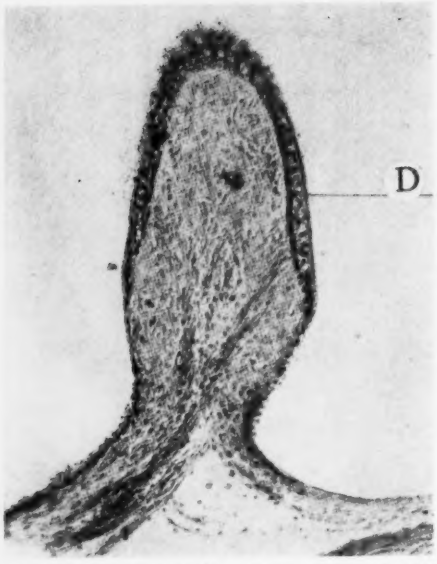


Fig. 3 (Animal 1408).—Crista. Neomycin 100 mg/kg/day s. c., 30 doses. Scattered degeneration of hair cells (*D*); × 215. (Reproduced with permission from Hawkins, J. E., and Lurie, M. H.: Ann. Otol. Rhin. & Laryng. 62:1128, 1953.)

followed multiple genitourinary reconstructive operations culminating in a relatively well-functioning left nephrostomy and a right cutaneous ureterostomy.

The past system and family histories were noncontributory except for rheumatic fever at 10 and 31 years of age.

Positive pertinent physical findings of this well-developed, well-nourished woman with a temperature of 37 C (98.6 F), pulse of 60, and blood pressure of 110/70 were as follows: A soft abdomen revealed a left nephrostomy and right cutaneous ureterostomy. Pelvic examination demonstrated a foreshortened vagina with considerable scarring and mucosal atrophy. The uterus was absent.

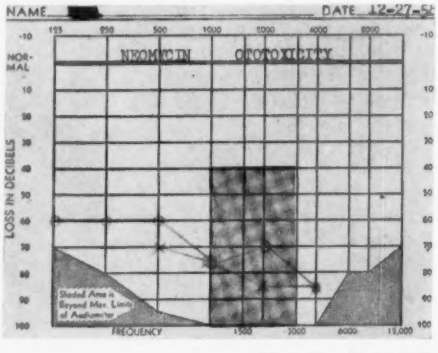
Pertinent admission laboratory data showed a hemoglobin of 15; urinalysis of the nephrostomy specimens revealed a yellow turbid specimen with a specific gravity of 1.008, pH 5.5, negative reaction to sugar, and a trace reaction to protein. Microscopically, many bacteria, 10 to 15 white cells, and an occasional red cell per high-power field area were seen. Differential PSP determinations revealed almost no renal function on the right but fairly good function on the left. The NPN was 50 mg. %; creatinine 2.4 mg. %. Blood type was O positive.

On Sept. 26, 1957, the first day after admission, a cystoscopy in surgery revealed the bladder to be small but of normal configuration. It could be distended to approximately 110 cc. Bladder distention regimen gave good results, and instilled fluids were held. On Sept. 28, 1957, the patient was put on a bowel preparation including oral administration of phthalylsulfathiazole (Sulfathalidine) 4 gm. (2 gm. q. 6 h. ×2) and neomycin sulfate, 11 gm. (1 gm. stat. and 1 gm. q. 6 h. × 4, followed by 1 gm. q. 4 h. ×6). On Sept. 30, 1957, under I. V. thiopental (Pentothal) and general intratracheal anesthesia, a left ureteroileoneocystostomy was performed. The procedure was moderately well tolerated and the postoperative course benign. Her postoperative chemistries remained within normal limits. However, after ambulation, upon clamping of the nephrostomy tube and removal of the urethral Foley catheter, incontinence developed, worse when lying down or walking. Examination revealed a vaginal fistula, either vesicovaginal or ureterovaginal; otherwise the patient was continent. She was discharged Oct. 30, 1957 to outpatient genitourinary clinic until further reparative surgery was deemed advisable.

On Nov. 5, 1957, the patient was readmitted to the U. C. L. A. Medical Center's Urological Service. Her physical examination was unremarkable, not differing from that previously reported.

After completion of her work-up the oral preoperative bowel preparation, including phthalylsulfathiazole 4 gm. and neomycin sulfate 9 gm. over approximately a 36-hour period, was again used. This was her second course of neomycin bowel preparation. After the ninth dose on Nov. 7, 1957, the patient vomited an estimated half of her last medication, possibly representing a toxic neomycin response. On Nov. 8, 1957, a left uretero-

Fig. 4.—Audiogram, Dec. 27, 1957, after nursing notation of Dec. 24, "Not hearing unless shouted at." Neomycin first administered Sept. 26, 1957, by mouth for bowel preparation. Second dose for the bowel preparation, Nov. 5. Third dose, Dec. 16, 1957, made up of 11 gm. intramuscularly followed by 5 gm. by mouth.



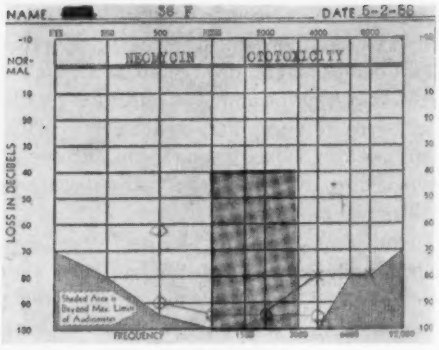


Fig. 5.—Audiogram of same patient as Figure 4, May 2, 1958, some five months and six days later, demonstrating further hearing deterioration.

ileosigmoid anastomosis was performed and a sigmoidal cystoplasty effected, followed by an uneventful postoperative 72 hours.

For the next surgical procedure of this admittance, it was thought best not to chance neomycin, so on Dec. 11, 1957, a second surgical procedure of converting the ileoureter into a ureteroileal-cutaneous anastomosis was done. Good urine drainage immediately occurred. However, on Dec. 13, 1957, incisional infection and pain coupled with fecal vomiting prompted taking a culture that on Dec. 13, 1957, revealed Pseudomonas aeruginosa and Proteus morganii, both sensitive to neomycin.

A summary of her laboratory findings is not pertinent to this report, except that as kidney function impairment increased (as shown by daily urinalysis for albumin, casts, and cell determinations) corresponding blood urea nitrogen and nonprotein nitrogen levels also rose, followed by continuing hearing deterioration. Her highest creatinine was 3.8 and NPN reached 90 on Dec. 27, 1957, the fourth day after neomycin therapy was stopped.

On Dec. 14, 1957, because of the increasing critical illness, neomycin sulfate, in recognized, successive increasingly large doses was given intramuscularly, a total of 16 gm. being given in nine days (11 gm. intramuscularly in four days followed by 5 gm. orally for five days). On Dec. 16, 1957, a dehiscence of the incision occurred in the right flank, followed by formation of a fecal fistula. Early general signs of toxicity were as noted, apprehension, depression, detachment, and restlessness, followed later by tinnitus of a sludging, fluctuating pitch, which was most distressing. Nurses' notes were further enlightening, such as Dec. 21, 1957, noting the patient's refusing her medication, stating it "made her sick," followed on Dec. 23, 1957, by increased ringing with some difficulty in hearing, this in turn giving way on Dec. 24, 1957, to vomiting; however, no dizziness was noted, in bed. Notations, including "Not hearing unless shouted at," "seems able to read lips to some extent," evidenced the rapid cochlear deterioration.

The patient's course was extremely stormy until the middle of February, when she improved as her NPN diminished to 38 mg. % and her serum electrolytes returned to normal. The fistula in the left lower abdominal quadrant had practically closed. The Bricker procedure appeared to have been successful. An air-conduction aid thought initially by the patient to be beneficial did not increase her discrimination. No recruitment was ever elicited.

Note: No audiograms were taken before neomycin medication. Audiograms after neomycin medication show loss of all serviceable hearing. For a comparison, audiograms, pre- and post-neomycin medication of one of Carr's[11] cases are shown. Subsequent Kobrak caloric tests failed to elicit any vestibular response. The patient, how-

TABLE 1.—*Results of Sensitivity Tests*
(Dec. 13, 1957)

	Neomycin	Polymixin B	Sulfisoxazole	Novobiocin	Sulfamethoxypyridazine	Nitrofurantoin	Trisulfapyrimidines	Tetracycline	Oxytetracycline	Streptomycin	Penicillin	Erythromycin	Chloramphenicol	Chlortetracycline
Right														
Proteus morganii	S	S	S	R	R	R	R	R	R	R	R	R	R	R
Pseudomonas aeruginosa	S	S	R	R	R	R	R	R	R	R	R	R	R	R
Left														
Streptococcus faecalis	—	—	—	—	—	—	R	R	R	R	R	S	S	R
Paracolobacterum coliforme	S	R	R	—	-	S	R	R	R	R	R	R	S	R
Escherichia coli	R	—	—	—	R	S	R	S	S	S	R	R	S	S

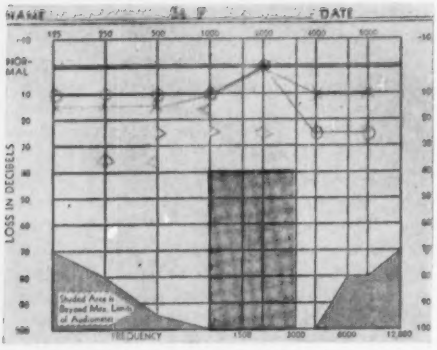


Fig. 6.—Audiogram of one of Carr's cases taken before neomycin administration parenterally for tuberculosis.

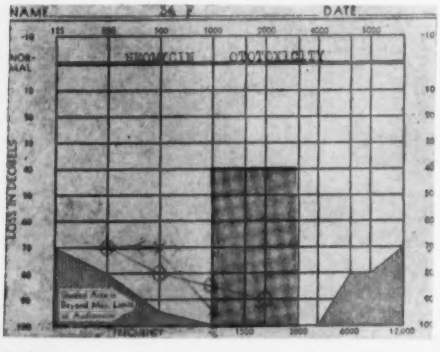
ever, cried from the intense pain produced by prolonging the icy water irritation in an effort to get some vestibular response.

The patient never complained of vertigo. She had been constantly bedfast in a two-bed ward on an active floor.

Precautions that may be emphasized previously to treating these critically ill patients, especially if they may have had or show any neomycin hypersensitivity, may be classified as general and otological.

General.—The patient must be hospitalized and be critically ill from bacteria resistant to other antibiotics or chemotherapy. Those responsible for the patient must be informed of the ototoxic potential of neomycin. Laboratory determinations, as urinalysis for albumin, casts, and cells

Fig. 7.—Audiogram of same patient as Figure 6 four months after parenteral administration, depicting the marked perceptive hearing deterioration.



every 12 hours, coupled with blood urea, nitrogen, and nonprotein nitrogen, as evaluation demands, are also required. Optimum neomycin concentrations effective to lyse the pathogen in vitro, are invaluable. Lastly, intradermal skin testing for neomycin hypersensitivity may be advantageously utilized.

Otological.—Inquire whether streptomycin or dihydrostreptomycin has been previously used, in order to obviate adding further irreversible Corti damage, it being felt that molecular changes with impairment of function precedes neuroepithelial damage as manifested clinically [18]; audiometric testing before, during, and after therapy may detect preclinical damage. If use of the audiometer is unavailable, then testing for the whispered and spoken voice, inquiring for sludging, high-pitched tinnitus, and vertigo is in order. The reliable tuning-fork tests of Rinne, Weber, and Schwabach for the speech frequencies 512-1,024 and 2,048 must be recorded. (Most clinicians have a 512 fork about.) If the patient's condition permits, it is judicial to catalogue water, electrical, or motion vestibular tests.

The term "sudden deafness" [25-31] was used in these collected case reports: Epstein has shown that from topically applied neomycin, a delayed dermal or tuberculin type neomycin sensitivity is manifested in the skin, this involving the hair follicle.[23] Both the skin and the inner ear are of ectodermal derivation. The thought is presented here that neomycin, having an affinity for the hair cells of the organ of Corti, might act as a mounting delayed tuberculin type of reaction to the organ of Corti hair cells, contributing to this "sudden deafness" terminology.

Streptomycin, dihydrostreptomycin, and neomycin, as shown in animal experiments and seen clinically in man, select different locations with correspondingly varying toxic affinities for the cochlea and vestibule.[12] That the toxicity of the "mycins" is not confined to the labyrinth has been shown by DeSa who has demonstrated the

TABLE 2.—*Parenteral Neomycin Ototoxicity*

Author	Age	Sex	Disease	Wt., Kg.	Dosage Parenteral Route	Dosage Parenteral Total	Duration of Therapy, Days	Cochlea Tinn.	Cochlea Deafness	Vestibule
Carr[11]	51	M	Tbc., pulm.	45.5	I. M.	37.0 gm.	34	..	Severe	Not recorded
	77	M	Tbc., pulm.	43.4	I. M.	34.0 gm.	41	..	Moderate	Not recorded
	41	F	Ca. ? uterus Tbc., pulm.	43.0	I. M.	45.0 gm.	42	..	Moderate	Not recorded
	34	F		61.6	I. M.	42.0 gm.	29	..	Severe	Not recorded
Kenoyer[19]	20	M	Pseudomonas endocarditis		I. M.	28.0 gm.	14	..	High freq. loss	Not recorded
Read & Wellman[21]			Staph. endocarditis		I. M.	21.0 gm.	35	..	Slight deaf. (in 5 wk.)	Not recorded
Bendove & Ashe[24]	79		Geotrichum septicemia (diabetic)		I. M. I. M.	1,000 U./kg. 500 U./kg.	11 {2 9	..	Impaired hearing	Persists
Powell & Hooker[25]	57	M	Bacterial endocarditis γ-Strep.		I. M.	19.0 gm.	19	..	Sudden	Not recorded
Waisbren & Spink[26]	66	M	Pseudomonas blood str. infection		I. V. I. M.	0.5 gm. I. V./ 12 hr. for 3 0.5 gm. I. M./ 6 hr. for 11	14	..	21 days after onset of therapy, hearing diff. in 12 mo. comp.	Not recorded
	18	M	Otitic meningitis; cerebellar abscess		I. V. I. S.	2 gm. 41.5 5 mg.	21	..	Impaired hearing	Not recorded
Kadison[27]	65		Lung abscess cardiac		I. M. or locally	54,000 U.	18	..	Complete	Not recorded
Duncan[28] 145 No. 2 1-13-51	48	M	Prostatic hypertrophy cystitis pseudomonas		I. M.	982,550 U.	3	..	Consid. impaired; (later 50% improved audiometrically)	Not recorded
Kadison[27] *	52		Tbc. knee		I. M.	21,600 U.	36	..	High freq. loss (later improved)	Not recorded
	19		Tbc. nephritis Tbc. cystitis		I. M. or locally	19,775 U.	58	..	High freq. loss	
Waisbren & Spink[26]	39	F	Perinephric abscess		I. M.	16.0 gm.	8	..	Complete	Function diminished
	67	M	Pyelonephritis		I. M.	8.0 gm.	4	..	Complete in 3 mo.	Function diminished
	59	F	Pyelonephritis		I. M.	10.5 gm.	7	..	Increasing	Function intact
	55	M	Pyelonephritis		I. M.	14.0 gm.	7	..	Progressive	Function lost at 4 mo.
	53	M	Carcinoma of bladder		I. M.	13.5 gm.	9	..	Complete	Function diminished
Greenwood	38	F	Carcinoma of uterus		I. M.	18.0 gm.	9	..	Complete	Function diminished

* Since this paper was submitted for publication, one more case of permanent deafness has been observed.

toxicity of dihydrostreptomycin involving the neuroglia of the acoustic pathway and auditory cortex.[29]

Pantothenic acid, one of the vitamin B components, as a calcium pantothenate, has been shown to lessen acute toxicity of the "mycins."[13] Report of a close physiological association of antibiotics and vitamins in abating stress is being noted. Here possibly four or five times as much vitamin C and pantothenic acid are utilized in accelerated adrenal cortical function.[20] Further investigation of the role of metal-binding agents lessening the toxicity of neomycin was recently reported.[30] However, the effective therapeutic potential was proportionately decreased as the toxicity diminished.

If this method permits the isotopic tagging of metals requiring electron transport enzymes, might not we have a means of investigating why the different "mycins" seek out and destroy certain cochlear and/or vestibular cell components?

Comment

As the topical use of neomycin increases, hypersensitivities are certain to mount. Where parenteral use is heroically employed, the symptoms and signs of early cochlear and vestibular toxicity must be recognized. Otologists must alert the attending medical and nursing personnel to the general and special precautions necessary to prevent hearing impairment. The general practitioner, surgeon, urologist, obstetrician, or gynecologist should know of these precautions. He must be aware that fullness in an ear and tinnitus are warnings of ototoxicity and that persistent neomycin medication in the presence of these may result in irreversible deafness, be it sudden or progressive.

Summary and Conclusions

Twenty cases of ototoxicity resulting from systemic use of neomycin sulfate are collected. An added case report corroborates the findings of the collected cases. Severe vestibular toxicity, as reported in two of the collected cases, was present here.

Some precautions in the systemic use of neomycin to obviate ototoxicity are presented. To this end attention is directed to reports of the synergistic antibiotic-vitamin pharmacological action and to the suggested role that isotopically tagged chelated metals might be microradiographically utilized in an effort to uncover why specific drugs affinitively attack certain cellular constituents of the labyrinth.

1033 Gayley Ave.

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The Ototoxicity of Kanamycin Sulfate in the Presence of Compromised Renal Function

RALPH F. NAUNTON, M.D., and PAUL H. WARD, M.D., Chicago

The potential ototoxicity of some antibiotics is well recognized, the most notable examples being streptomycin,[1] dihydrostreptomycin, and neomycin. Our recent experience with two cases, who suffered eighth nerve damage following the administration of kanamycin sulfate (Kantrex[2]), is presented as a reminder of the risks involved and as an illustration that these risks may be much increased in the presence of compromised renal function.

CASE 1.—A 40-year-old white male paraplegic, a veteran of World War II. His general health had been excellent until a shrapnel injury in 1945 resulted in total paraplegia. He was successfully rehabilitated after four years of hospitalization and has been able to drive his own car and operate a successful accounting business. A cordotomy was performed in 1956 because of increasingly severe burning pain in the lower extremities. After six weeks the pain recurred, and the patient has since become addicted to dihydromorphinone (Dilaudid). From 1956 to the present time (July, 1958) he has been hospitalized many times for "a bleeding esophageal ulcer," anemia, and recurrent attacks of cystitis.

Nine months prior to his recent hospital admission intravenous pyelograms revealed a hydronephrosis of the right kidney and a markedly dilated and kinked right ureter. Attempts to correct the kidney defect were unsuccessful, and a right nephrectomy was performed. Six months later a transurethral resection was carried out to insure adequate urinary drainage because the patient had a successfully trained, manually operated bladder.

One month before admission to the University of Chicago Clinics a urinary infection, with a temperature of 105 F, led to the patient's admission to his local hospital. Pseudomonas organisms were cultured from the urine—an infection that failed clinically to respond to methenamine mandelate (Mandelamine), chloramphenicol, (Chloromycetin) penicillin, and nitrofurantoin (Furadantin). At this time the patient's blood urea nitrogen was 50 mg/100 ml. Intramuscular kanamycin sulfate was started at a dosage of 1 gm. twice daily, and the fever subsided. On the seventh day of kanamycin sulfate administration the patient developed nausea, vomiting, vertigo, tinnitus, and a rapidly progressing deafness. By the eighth day he was totally deaf. Kanamycin sulfate administration was immediately stopped, a total dosage of 14 gm. having been administered.

Tinnitus, vertigo, nausea, and vomiting persisted for two weeks after discontinuation of the kanamycin sulfate but have since gradually subsided. The blood urea nitrogen reached a maximum of 126 mg/100 ml., but the urinary output was always above 1,500 ml. At this point the patient was admitted to the University of Chicago Clinics for possible "artificial kidney dialysis." Proteus and Pseudomonas organisms were cultured from the urine, an infection which has since been brought under control by sulfisoxazole (Gantrisin). Hearing tests carried out soon after the patient's admission to this hospital and again more recently have shown a total bilateral loss of hearing; tests of vestibular function have demonstrated a profound but not quite total loss of activity on both sides.

CASE 2.—A 48-year-old white man, who was seen in our clinic complaining of deafness of eight months' duration. In August, 1957, he had a right nephrectomy for a renal malignancy. He subsequently developed an infection in the remaining kidney and was admitted to his local hospital in November, 1957, where he received 1 gm. of streptomycin sulfate daily for 14 days. On the third day of streptomycin therapy he noted a "buzzing" in his right ear, which heralded a gradual decrease of the hearing in this ear. The patient claims that until this happened his hearing had been excellent and that the left ear appeared to remain unaffected at this time. Seven months later, in June, 1958, his urinary tract infection recurred. He was hospitalized again and given 1 gm. of kanamycin sulfate intramuscularly each day for five days, a total dosage of 5 gm. The patient states that at this time the hearing in the "remaining" or left ear diminished to a point where he had profound difficulty in hearing speech.

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From the Section of Otolaryngology, The University of Chicago Clinics.

A clinical history taken in July, 1958, during an outpatient visit to the University of Chicago Clinics made to discover whether "anything can be done about my hearing," revealed the points recounted above; further questioning failed to suggest any possibility of preexisting auditory trauma or other ear disease, or of a familial tendency towards deafness. Physical examination at this time revealed a scar of the right eardrum but was otherwise unremarkable. Hearing tests showed a total loss of hearing in the right ear and a profound nerve deafness (average of 65 db.) in the left ear. Tests of vestibular function revealed diminished activity on the left side only, the activity on the right side being grossly normal.

Comment

These tragic cases may serve to illustrate two important points that should be borne in mind concerning the clinical administration of kanamycin sulfate: 1. Our admittedly limited experience with this drug has indicated that it can, even when used in small doses, damage or destroy the hearing mechanism. The clinician contemplating the use of the drug must decide whether such serious risks are justifiable. 2. The potential ototoxicity of kanamycin sulfate, and of other ototoxic antibiotics excreted by the kidney, may be greatly enhanced by compromised renal function.

Summary

Two similar cases are presented in which severe eighth nerve damage followed the administration of relatively small doses of kanamycin sulfate. One case received 14 gm. over a seven-day period and the other case received only 5 gm. in a five-day period.

Each case had had a previous nephrectomy and was thus in a state of compromised renal function despite adequate urinary output.

It is emphasized that new drugs with ototoxic potential should be used with the greatest caution, particularly in cases with questionable renal function.

950 E. 59th St. (37).

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A Method of Otoplasty*

Normal Contour of the Antihelix and Scaphoid Fossa

RICHARD T. FARRIOR, M.D., Tampa, Fla.

A technique for the correction of protruding ears is presented. Based on a study of cadaver sections and anatomic observations of the "average normal" ear, the procedure attempts to duplicate as nearly as possible the contour of the ear as seen in the lateral view. There are many procedures for the correction of protruding ears; in our hands the technique presented more nearly simulates the normal in contour.

It is not the purpose of this paper to review previous techniques or all the details of the abnormal anatomy of the protruding ear; however, certain methods which have lead to the present procedure and pertinent anatomical details will be stressed.

It is interesting that neither in the clinical literature nor in the standard anatomy text has there ever been a description of cross sections of the auricular cartilage. Many artists' concepts are included in the literature, and the topographical anatomy is quite adequately described. The cross sections indicated in Figure 1*A* and shown in Figure 1*B*, showing just what it is the surgeon is attempting to duplicate, are the first such sections published as well as I have been able to determine. I would like to suggest that such sections be included in the training program for all residents intending to do otoplastic surgery.

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* Illustrated Article, shown as a Scientific Exhibit at the Florida Medical Association Convention, Miami Beach, May 10, 1958, and Southern Medical Association Convention, New Orleans, Nov. 3, 1958.

I have made drawings of these sections in Figure 1*C* which may be compared with the abnormally protruding ear in Figure 1*D*.

In the normal the superior crus effects a gradual convexity blending with the shallowing scaphoid fossa and with the triangular fossa on either side. In the protruding ear the convexity is lost and may even become a concavity. The inferior crus is acutely convex and most frequently is not altered in the protruding ear.

The horizontal section across the concha, antihelix proper, and scaphoid fossa demonstrates a very definite and prominent antihelix with the auricular cartilage making a horseshoe turn upon itself. The scaphoid fossa is a distinct groove between the antihelix and the helix. Again the convexity is lost in the protruding ear. The fact that the cartilage is flat or concave is ignored in the surgical techniques which simply break the cartilage with a single incision. There may be actual cupping forward and the conchal cavity is often abnormally deep. The auriculomastoid angle usually is not altered, although, as mentioned, the conchal cavity may be abnormally cupped. It is interesting that in this section the cartilage does not extend into the helix as it is so often drawn.

Figure 2 is a composite of previously published techniques which are frequently used. The antihelix, especially in the superior crus, may be too sharp; there may be overriding or underriding of the cartilages apparent from the anteriolateral surface, or the scaphoid fossa may be lost or flattened by simply tipping the helix backward. Where the overriding cartilage strip is utilized a fairly satisfactory antihelix may result in the inferior section;

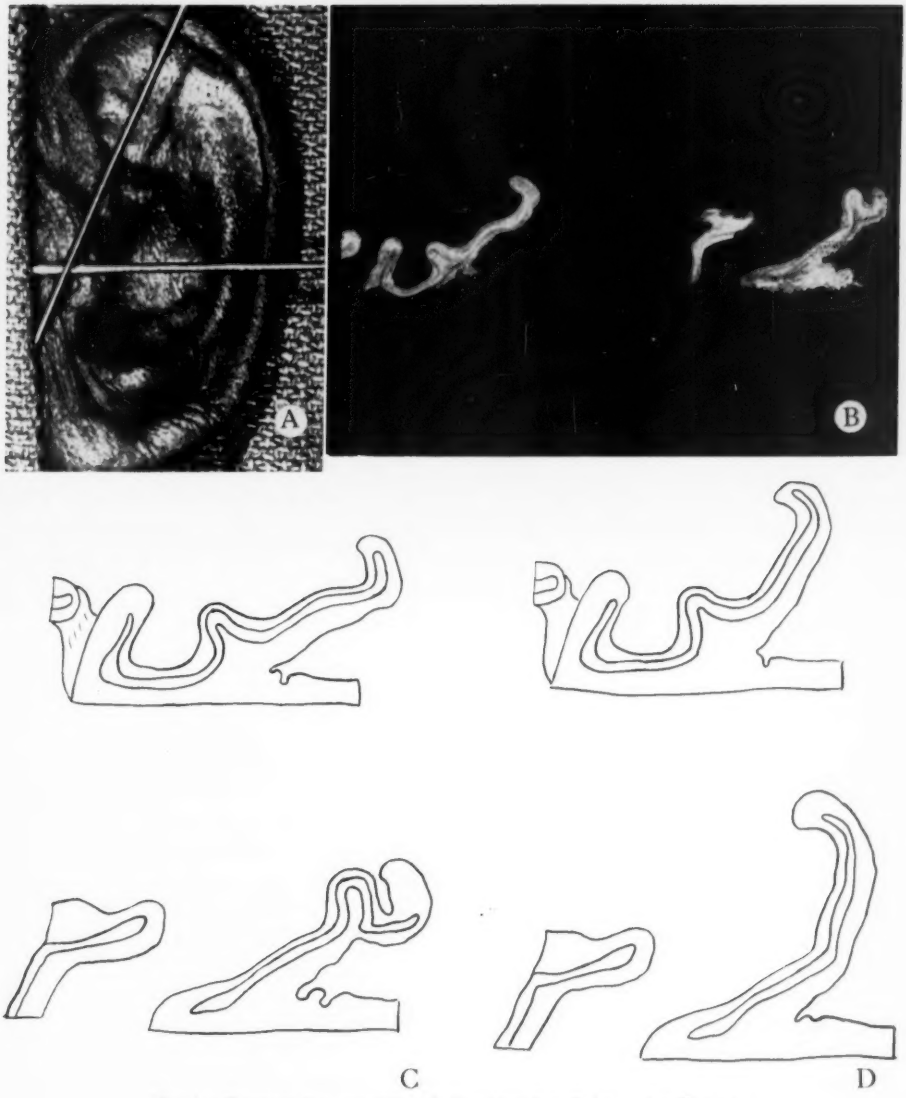


Fig. 1.—Comparative anatomy of the normal and the protruding ear.

however, superior to this and particularly at the superior crus, the strip is too narrow and prominent. In the lower drawing the normal is more nearly approached; however, the superior crus is flat, and the incisions through the thickness of the cartilage may be seen on the anteriolateral surface.

Becker [1,2] and Converse [3] have in their techniques been conscious of most of the points mentioned and have produced excellent appearing ears and, I am sure, many happy patients. I quite humbly submit what I feel are the shortcomings of their techniques as published. In each of their techniques incisions are made through the cartilage about the entire antihelix. In my experience these incisions are visible through the skin, making a sharp line on the anterior surface. There is a continuous

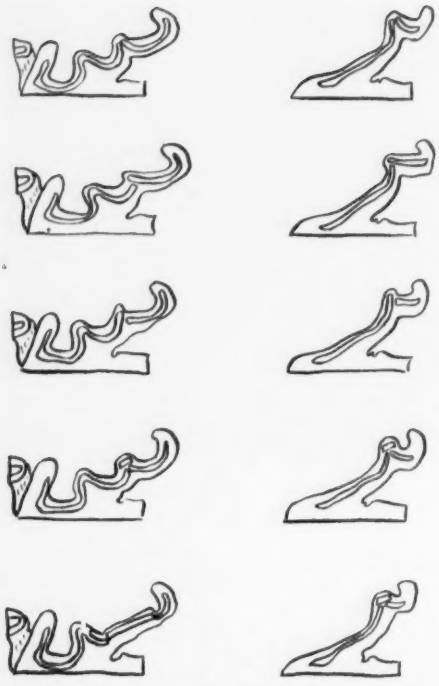


Fig. 2.—Summary of results of other operative techniques.

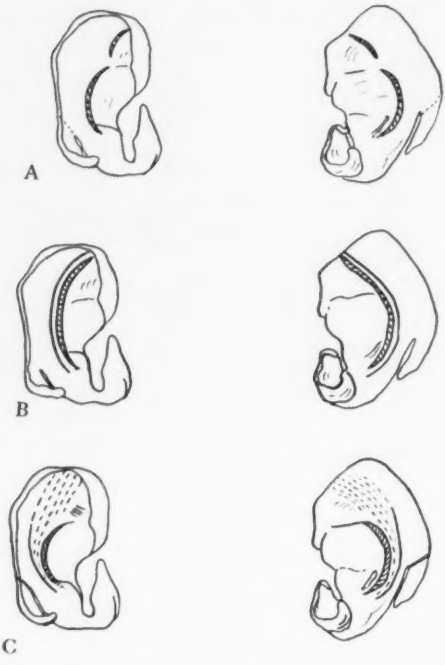


Fig. 3.—Comparison of previous techniques with present technique.

incision from the triangular fossa margin of the superior crus to the antihelix proper about the cavum conchae. With this incision there is a loss of the natural convexity at the junction of the two crura with the antihelix proper.

The use of the wire brush for thinning the cartilage has an effect similar to the technique I am advocating, although the principle was arrived at independently. I have used the smallest dermabrader to remove parallel concave troughs similar to the wedges removed in the current technique. I feel that the strips of full-thickness cartilage which remain between the troughs give body and resilience to the ear.

Straith [13] now does not join the superior crus and conchal rim incisions as he describes in his article, but leaves an isthmus of cartilage at the junction of the crura (Fig. 3*A*). This effects a more pleasing contour and eliminates the prominent ridge of cartilage and acute angulation in this area. This principle is utilized in my technique. Dr. Straith's technique, as shown in Figure 3*A*, produces a sharp anterior margin of the superior crus, and there is no attempt to make a prominent antihelix proper with the resulting scaphoid groove.

Huffman [6] was using the method illustrated in Figure 3*B* in 1952. The method of the three incisions and an overriding cartilage strip is utilized by me in the reconstruction of the antihelix proper. Superiorly the strip is too narrow and does not produce the desired slow convexity of the superior crus. There is often some flatness posterior to this too acutely convex superior crus. There is no break at the junction of the crura with the antihelix proper.

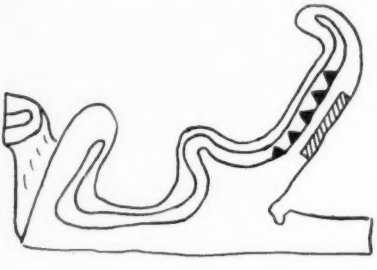
The last two procedures I mention in particular, as both authors were former instructors and I am much indebted to each of them. Many principles they taught are embodied in my own technique and each

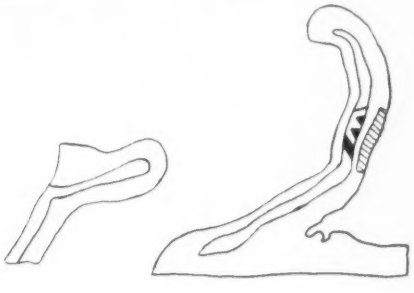
method was a forerunner of the one I am now using.

Present Technique

Figure 3*C* shows in diagram my present technique. In brief, incisions are made through the cartilage only about the conchal rim, extending slightly along the inferior crus, and paralleling the scaphoid fossa to the level of the junction of the crura with the antihelix proper. At this level the outer incision becomes superficial, and only a longitudinal wedge of cartilage of decreasing depth, not extending through the full thickness, is removed. The dotted lines depict the area where longitudinal cartilage wedges only are removed. There are no incisions through the cartilage about the superior crus. The strips are rounded superiorly to give a more pleasing curve. Care is taken not to extend too far toward the helix rim as undesirable angulation results. There is a gap between the incision about the concha and the removed cartilage wedges at the area where the crura join the antihelix proper. Parallel longitudinal wedges are removed along the entire antihelix to produce the desired convexity from the anterior surface. The amount necessary for the individual ear is removed as a strip of cartilage about the conchal rim.

Figure 4 is drawn from the same cross section across the superior and inferior crus, and shows the technique for producing the gradual convexity of the superior crus which blends with the triangular and scaphoid fossa on either side. In removal of the cartilage strips care is taken not to extend through the full thickness of the cartilage. It is usually not necessary to remove cartilage in the area of the superior crus. It is necessary to remove only the redundant skin on the posterior surface which results from bending the ear into normal position. The slight tension resulting helps to retain the cartilage in the desired position.





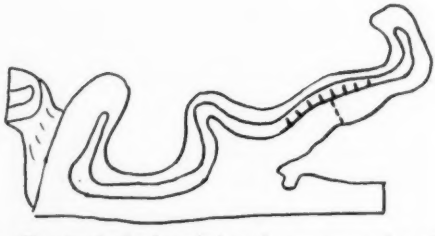


Fig. 4.—Author's technique for reconstruction of the superior crus.

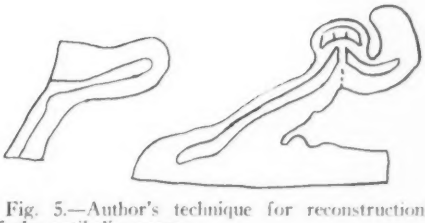


Fig. 5.—Author's technique for reconstruction of the antihelix proper.

Figure 5, drawn from the horizontal sections across the concha and antihelix, demonstrates the removal of the cartilage wedges continuing into the antihelix inferiorly. The incisions through the cartilage on either side are beveled to produce a better dome from the anterior surface. The cartilage strip remaining in widens progressively from the inferior extent to the junction with the crura. A strip of cartilage the required width is removed about the conchal rim. The overriding cartilage strip widens superiorly to blend with the superior crus. Sutures may be placed at the edges of this overriding cartilage similar to those used by Dr. Converse to increase the convexity. These sutures usually are not necessary. The underriding cartilage is brought together with horizontal mattress sutures.

The Surgical Procedure (Figs. 6*A* through 6*I*).—Figure 6*A*: The incision is made on the posteriomedial surface of the auricle opposite the antihelix. The auriculomastoid angle is preserved with this incision. Although the excised skin is generally elliptical in shape, I would emphasize that in the midportion and for the greater extent of the excision the two incisions are parallel or even slightly closer together than at either end. This prevents the pinched-back appearance which is seen with some operations in the central one-third of the ear. If the surgeon is not certain how much skin should be excised, only the outer incision is made initially, and the skin dissected back as a flap. The necessary amount of skin may then be removed at the end of the procedure.

Fig. 6.—The surgical procedure (see text).

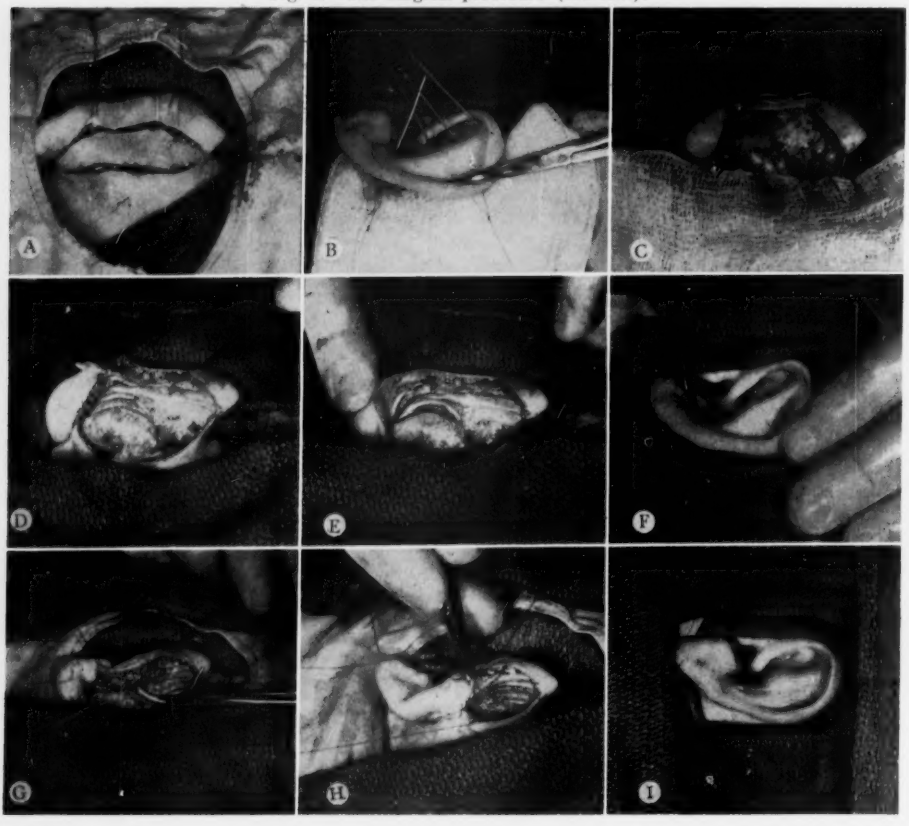


Figure 6*B*: Utilizing straight cutting needles, only four points are marked on the anterior surface. From above downward these are (1) the triangular fossa margin of the superior crus beneath the helix; (2) the triangular fossa margin of the superior crus at the junction with the inferior crus; (3) the conchal rim at the junction of the antihelix proper with the inferior crus; (4) the conchal rim at the inferior extent of the antihelix. No dye is used.

Figure 6*C*: With the skin dissected off the posterior surface the landmarks are readily visible. The needles from the anterior surface serve as guides. A scratch only is made over the needles into the cartilage along the margin of the triangular fossa, and another about the conchal rim. There is a break between these two lines at the junction of the crura. The needles are then withdrawn, and it has not been necessary to stain the cartilage, skin, or Mayo table with dye.

Figure 6*D*: The incisions described above have been made and the cauda helicis exposed. The strip of cartilage to be removed about the concha has been outlined with incisions through the full thickness of the cartilage. The longitudinal cartilage wedges have been removed. A fresh knife blade only is required for this maneuver. For this step I have used the No. 15 and the Lang, Bard-Parker blades or the No. 8 blade for the No. 44 Beaver knife handle. The round knife blades help prevent extension through to the anterior surface; however, the point of the No. 15 blade is sometimes utilized to remove the wedges from their trough.

Figure 6*E*: Here the cauda helicis and the excess conchal cartilage have been removed. The cartilage strip from the concha in this case was wider than usual, owing to the unusual depth of the cavum conchae in this patient. Frequently it is necessary to remove only a 1 mm. strip, and this is often done simply to insure a definite break in the cartilage spring. The incisions about the antihelix proper are beveled to make a smooth dome from the anterior surface. The cauda helicis is removed only when it is contributing to the prominence of the ear.

Figure 6*F*: The ear is pressed into normal position to demonstrate that the cartilage spring has been broken along natural folds. The ear is examined for any undesirable projections or irregularities as seen from the anterior surface and lateral view.

Figure 6*G*: The cartilage which is to underride the reconstructed antihelix proper is sutured with horizontal mattress sutures. Usually only three sutures are used, and it is not necessary to suture above the junction of the crura. At the present time I am using a 5-0 braided white Dacron suture. The suture is passed well into the substance of the cartilage to insure a firm grip. This step is felt to be necessary in order to produce an adequately prominent antihelix and as added insurance for maintaining the cartilage in the new more nearly normal position.

Figure 6*H*: The skin is closed with a continuous subcuticular suture of 4-0 monofilament nylon with an atraumatic cutting needle. The suture is walked to the surface in the middle of the incision to facilitate removal. Not only does this suture minimize scar, but the ease with which it is removed in children makes it worth while.

Figure 6*I*: The appearance of the ear following all suturing is shown. Note the slow curve or only slight convexity of the superior crus, the prominent antihelix proper, and the definite scaphoid fossa. The superior crus is subtle, yet the angulation adequate. The ecchymosis resulted from a slight undermining of the cartilage strip remaining in, done to allow it to assume its new position. These operative photographs are taken of the girl shown in Figure 7.

The Surgical Results.—Figure 7*A*: This attractive young lady was quite sensitive about her prominent ears. She is the person whose operation is shown in Figures 6*A* through *I*. The detraction from her generally attractive appearance produced by the prominent ears is apparent. She was

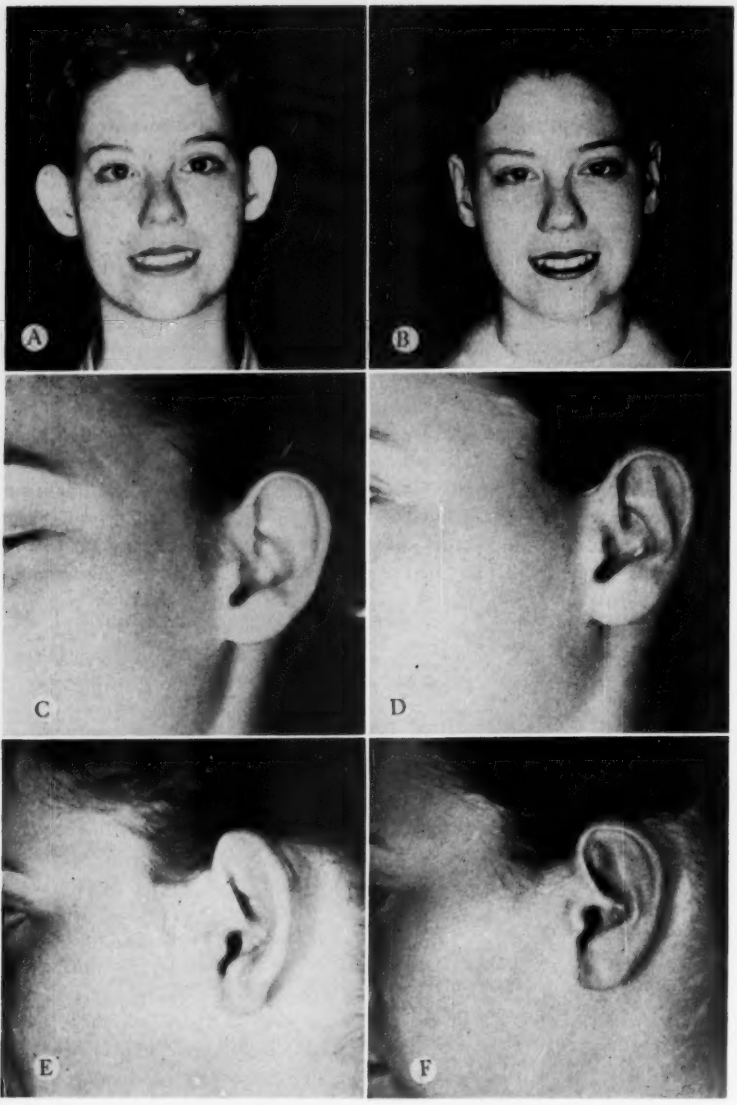


Fig. 7.—Operative results.

self-conscious and careful about her hair style.

Figure 7*B*: With the procedure described the protrusion is satisfactorily corrected without distortion by over- or undercorrecting in certain areas. Normal angulation from the side of the head is preserved. The psychological improvement with this patient was most gratifying.

Figure 7*C*: In the oblique view the abnormalities previously described are emphasized. Note the loss of the antihelix convexity and scaphoid groove. The cavum conchae is abnormally deep.

Figure 7*D*: Postoperatively the gradual convexity of the superior crus without visible incision lines or angulations is demonstrated. The antihelix proper is subtly

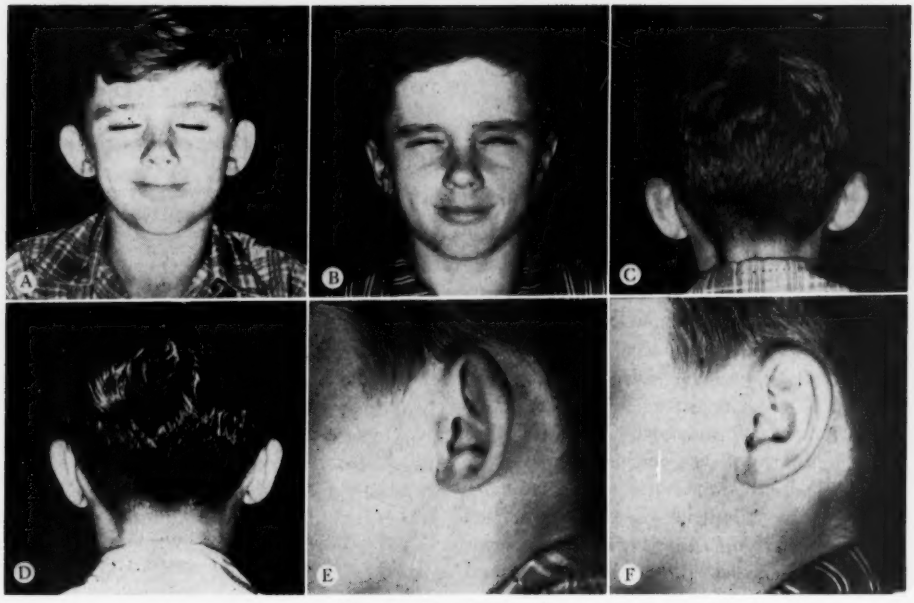


Fig. 8.—Operative results.

prominent, and there is a definite groove between the antihelix and the helix.

Figure 7*E* and *F*: The pre- and postoperative lateral views show the corrected angles, and again I would emphasize normal contour as seen from the side.

Figure 8*A* and *B*: The pre- and postoperative anterior views of this young man show satisfactory correction of the protrusion.

Figure 8*C* and *D*: Going away, the corrected protrusion is again demonstrated. Maintenance of a normal auriculocephalic angle without obliteration of the sulcus is accomplished. The incision, closed with a continuous subcuticular suture, is barely visible.

Figure *E* and *F*: Pre- and postoperative lateral views again are emphasized. The contour of the reconstructed antihelix and scaphoid fossa quite nearly duplicates the normal.

Summary and Conclusion

The objective of the technique presented is to simulate the normal folds and crevices of the ear in the lateral view as well as to simply correct the protrusion. A study of the "average normal" ear has been made by cadaver sections, and the abnormal protruding ear compared.

Line drawings made by me directly from the cadaver sections are used to compare the normal and the abnormally protruding ear. Using these same sections, my surgical technique is briefly described.

Details of the surgical procedure are correlated with photographs taken during surgery. Pre- and postoperative pictures of the patient operated on in the surgical photographs are shown.

In each of the postoperative pictures the appearance of the ear from the side view is emphasized. The technique has on occasion been combined successfully with the Kolle-Lexer and a modified Cocheril procedure for reducing the size of the ear, particularly in unilateral macrotia.

In conclusion I would like to emphasize these details of the technique:

1. Only the amount of skin necessary for effecting satisfactory angulation and eliminating redundancy is removed. This is

taken opposite the antihelix on the posteriomedial surface, and the auriculomastoid sulcus is preserved.

2. No dye is utilized in the skin or cartilage for identification of landmarks and outlining the incisions.

3. No incisions through the full thickness of the cartilage are made about the superior crus to the level of the junction of the two crura with the antihelix proper. In this area and extending into the antihelix proper parallel longitudinal wedges of cartilage are removed. Careful removal of the cartilage strips without extending through the full thickness is required.

4. A definite break both in the incisions and removed cartilage wedges is left at the junction of the crura to allow a more natural curve. The continuous incision from the triangular fossa margin of the superior crus to the conchal rim is discouraged and not used in this method of otoplasty.

Bayshore Blvd. at Hyde Park Ave.

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A New Procedure for Correcting Outstanding Ears

EDGAR M. HOLMES, M.D., Boston

An ear possesses individuality of its own, no two being exactly alike. Ears can identify persons nearly as accurately as their faces. The majority of persons have ears which fall within certain mathematical measurements as to size and shape and are considered as normal. There are those which are smaller and misshapen, which extend down the scale to the rudiments of an ear which we call a "microtic ear." On the other end of the scale, we have ears that stick out from the head because they do not have all the folds of a normal ear and because of this are more prominent and are called a "lop" or "prominent" ear. Some of this group are not only more prominent but are cupped, and a few are large in size. It is the operative alteration of the prominent ear that is to be discussed in this paper.

The auricle obtains and maintains its shape from its skeleton, the cartilage. Cartilage is nearly a perfect spring in that it may be bent for long periods of time, but once the force which holds it is released it will spring back to its former shape. Because of this inherent characteristic, any procedure which is to alter the shape of the auricle by bending must first remove the temper of the cartilage before it will remain in its new position. All successful procedures utilize this principle. They differ only in the way in which it is accomplished.

The majority of ears are outstanding because the antihelix has not folded backward, and it is the creation of this fold which is the crux of any practical procedure. When examining these cases before operating, one can usually hold the ear back into the position desired by digital pressure on the helix; this will create a bending of the antihelix and show the operator the shape which he would like to create. If the skeleton of the ear were composed of a malleable metal, this is all one would have to do as there would be no spring to return it to its former position. Because of the nearly perfect spring of cartilage, earlier operations which removed postauricular skin, even to the extent of obliterating the postauricular sulcus, failed. In time, the constant pull of the cartilage would stretch the postauricular skin and scar out to nearly its old position. It is the method of breaking this spring so that the ear may be molded to its new position without creating ugly creases or prominences on the anterior surface of the ear which has perplexed the surgeon over the years.

A review of the literature reveals that each new technique attempts to shape the auricle by breaking this spring and at the same time tries to avoid the creation of undesirable irregularities which may show on the anterior surface. Luckett [1] presented the technique of making cuts through the cartilage along the line on which he wished the cartilage to fold. This did help but was prone to leave an angulation instead of a general, rounding contour. This angulation in certain cases was rather conspicuous even

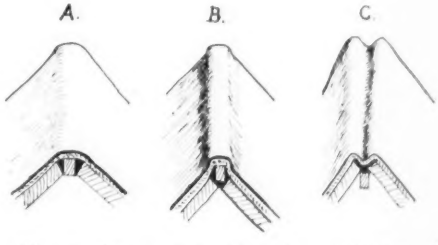

Fig. 1.—Diagrams indicating principle of Luckett's technique, variations of which are widely used. *A*, ideal curving of skin over cartilage. *B*, Undesirable result. Strip of cartilage pushed out, leaving a ridge. *C*, Undesirable result. Strip of cartilage pushed in, leaving a valley.

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to the uneducated eye. Later on, two and three parallel cuts were made in the hope of obviating that, but even these sometimes would buckle and leave their undesirable mark which was prominent beneath the skin (Fig. 1*A*, *B*, *C*, and Fig. 6). Further effort to obviate this undesirable condition was forwarded by Dr. McEvitt,[2] who made two parallel cuts, removed the cartilage between them, undermined the skin on the anterior surface of the cartilage, and by suturing the edges together had a redundancy of the skin which was to cover over the sharp bend of the cartilage and thus camouflage it. Even this was not satisfactory in all cases. Dr. John Converse et al.[3] presented a still further improvement in technique. He formed a cone of the cartilage, which created an antihelix with its apex end at the bottom. This was a marked advance. The results which he presented were unusually good. Technically, I believe this is one of the better methods presented so far.

Over the years, I have tried various methods and a few years ago was using multiple, parallel, oblique cuts through the cartilage, directed at about a 45 degree angle along the fold of the antihelix which we wished to create. An endeavor was made not to go through the cartilage on the anterior surface, but this was not always possible as it is difficult to cut partially through cartilage of various thickness without occasionally coming to the surface. When this occurred, an irregularity of the anterior surface would present itself. The principle of this was to have the anterior surface of the

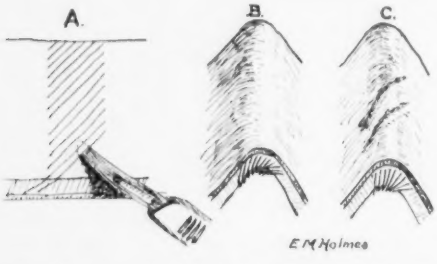

Fig. 2.—*A*, parallel tangential cuts through cartilage diagonal to desired bend. *B*, ideal gradual bend. *C*, two cuts extending through cartilage, creating sharp ridge across fold.

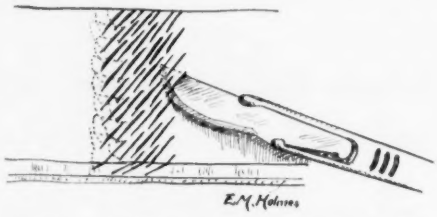

Fig. 3.—Cartilage spring broken employing multiple, diagonal interdigitating cuts. The posterior surface well cut, the anterior surface barely pierced.

cartilage a continuous sheet but its thickness to the posterior surface cut so that each little piece would be held in place on the anterior surface and yet slide to a new position beneath it when bent. When this was healed via scar tissue, the ear would then maintain its new position, all spring having been broken (Fig. 2*A*, *B*, *C*; Fig. 7*B*).

Because certain of these cases presented irregularities of the anterior surface which were disturbing to me, I conceived the idea of making shorter cuts by pushing a knife blade from behind through the cartilage to the anterior surface. This would give a wedge-shaped cut with its base posteriorly and the apex near or just through the anterior surface. The first row of cuts would be made along the most posterior portion of our desired bend of the antihelix. The next row would be parallel to it, and each cut would interdigitate with the preceding ones but not connect with them. The third row would interdigitate with the second, and in this way the entire area which we wished to bend would be cut. Where the cuts were tangential and overlapping, the cut strips would slide by one another. With no long cut on the anterior surface, bending was possible, but frequently the posterior surface had to be scarified to further break its continuity and, thus, its spring. Once the spring is broken sufficiently so that the ear will stay in its new position without being held, it may then be sutured in the desired position by through-and-through mattress sutures. This method was a big advance and one I employed for four years (Figs.

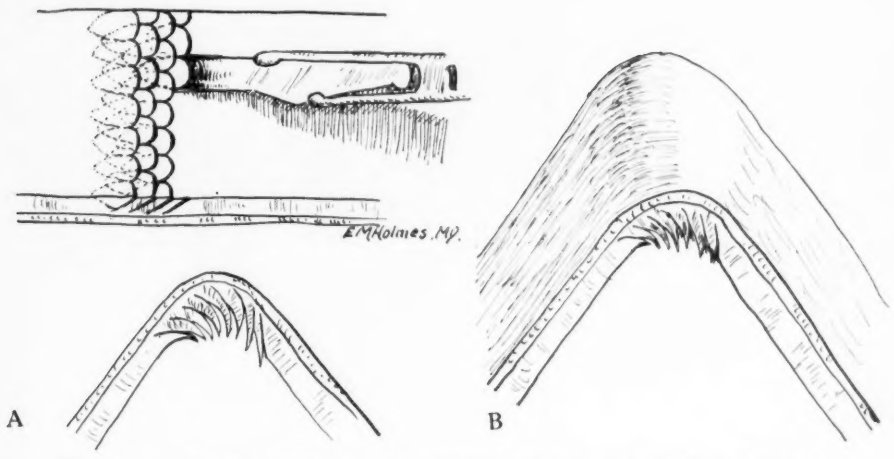

Fig. 4.—*A*, method of breaking cartilage spring using my special gouge. Point of blade should come just through cartilage on anterior surface. Each cut should overlap and communicate with each other on the posterior surface. *B*, ideal bend without irregularities of skin.

3 and 8). Every now and then a case would displease me because of a sharp ridge showing up across the antihelix.

Seeking perfection, what other technique could effect our desired result? Thinning the cartilage would work as shown by Dr. John Converse,[3] but I wanted something which would leave the main bulk of the cartilage attached to the thin anterior surface so that when healed scar tissue would create a bond and hold it in its new position. In nature, the skin of a fish or snake with its scales is an excellent example of what was wanted. To create this in cartilage would be nearly impossible, but, even if it could be approached, the solution might be solved. A special gouge was constructed and tried on a piece of septal cartilage. It worked beautifully not only on septal cartilage but on auricular cartilage in situ, so it is the method now employed by me for breaking the spring in prominent ears (Figs. 4 and 9).

The operation for a prominent ear is now conducted employing the following procedures. An incision is made about ⅛ in. laterally to the postauricular fold from its top to its bottom. The skin is then dissected from the posterior surface of the auricular cartilage until the entire area which is to be worked upon is exposed. This occasionally includes the helix so that the entire posterior surface can be visualized at once.

When the extent and degree of bending of the antihelix is determined, the area involved has its spring broken using my latest technique. The area is either outlined by marking the boundaries with methylene blue or if one is familiar with the reverse anatomy of the posterior surface of the auricle, he proceeds without the need of artificial markings. It is important that the cartilage be freed of all soft tissue as it is difficult to control cuts through cartilage if one must first cut through muscle, perichondrium, etc. Employing the special gouge, the first row of cuts is made obliquely to the posterior surface of the cartilage along the anterior or posterior limits of the area to be bent. One turned-up edge of the first cut coincides with the proximal edge of the next cut. When a row of cuts has been made, this simulates a row of scales. The point of the blade is pushed through the cartilage until it just reaches or goes through the anterior surface. This can be estimated by the feel of the instrument or by a finger on the skin over the front surface. The next row of cuts is parallel to the first and near enough to it so that the

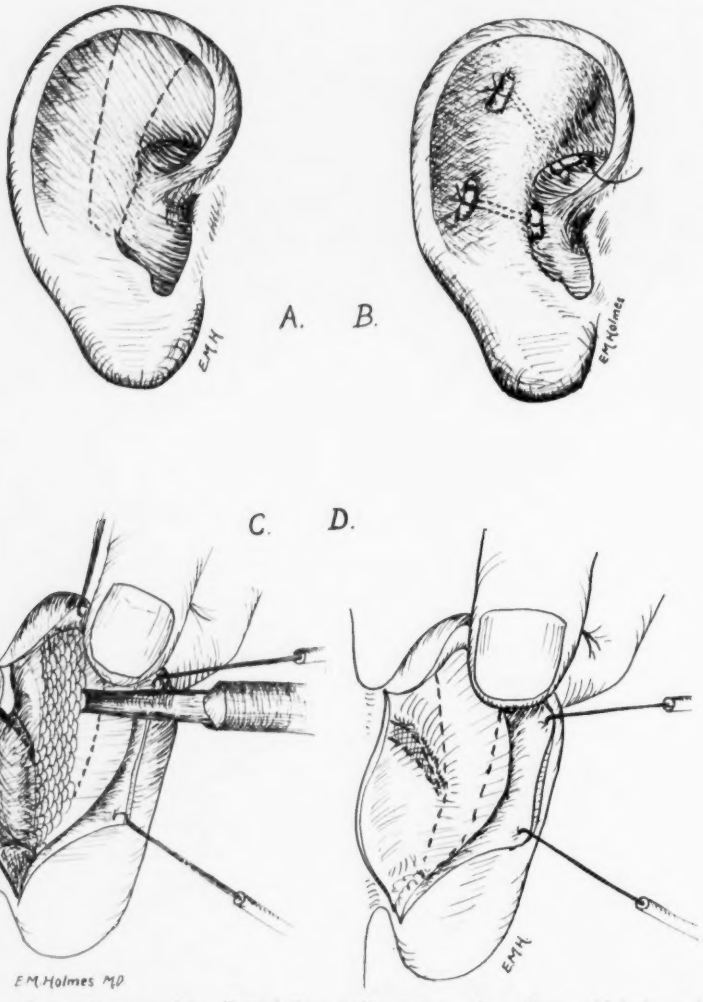

Fig. 5.—*A*, prominent auricle. Dotted lines indicate area of cartilage which is to have spring broken. *B*, stay sutures. Arrow points to area where cartilage may be cut through or excised without showing on anterior surface. *C*, exposure of posterior surface of auricle and cartilage being scaled. Note area in which conchal cartilage may be excised without showing on anterior surface (arrow). *D*, exposure of posterior surface. Dotted lines indicate area to have spring broken. Cross-hatched area, cartilage which may be removed without being seen on anterior surface.

bent edges of the blade come through the middle of the first cuts. In this manner, each cut of the second row overlaps those of the first, creating a pattern as scales of a fish or shingles on a roof. Row after row is made until the entire area that is to be bent is covered (Fig. 4 and Fig. 5*A*, *C*, *D*). This breaks the spring of the flat surface which is to be bent.

There are several other procedures which may be employed to overcome other problems which arise in various ears, but the above-mentioned procedure is all that is necessary in many cases. In some cases, the tissue between the concha and the mastoid cortex is removed, thus permitting the concha to fall nearer the head. This in itself materially helps in correcting some

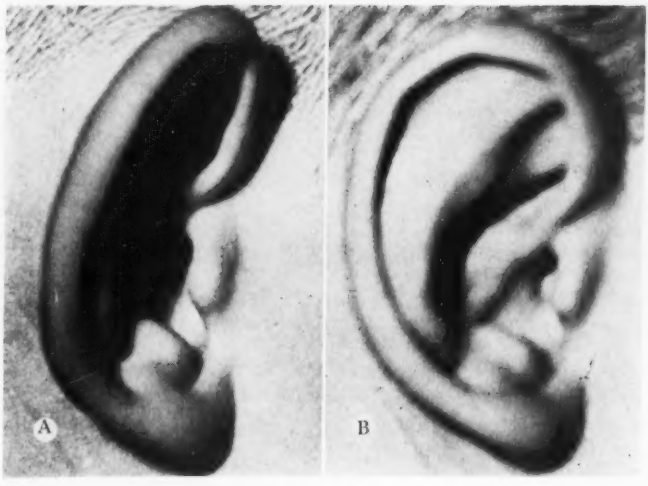

Fig. 6.—*A*, preoperative ear.* Note slight formation of antihelix and cupping of the lower third of auricle. *B*, demonstrates sharp bends in the cartilage using multiple cuts through the cartilage parallel to the desired bend of the antihelix. The result is good but the sharp folds are undesirable.

ears and is all that need be done in a prominent ear that has a well-developed antihelix. In a certain percentage of these ears, further correction which may be needed can be obtained by planing off the base of the conchal cartilage. Several degrees of prominence may be corrected in this manner.

Occasionally the helix itself must have its spring broken in several places; a strip of cartilage may have to be removed from the concha beneath the crura of the antihelix; the cordate process may need cutting across or even removal in some cases. The lobule may need splitting parallel to its outer surface, sliding the two surfaces to make it rest in a new position. These and other procedures must all be known and used to advantage when indicated. When the spring has been sufficiently broken, the auricle will nearly remain in its new position without holding it there. To make certain that it will not change shape while the pressure dressing is applied, through-and-through stay sutures are inserted. These are placed

* In illustrating this paper, an attempt was made to choose ears which were of similar formation before operation. In this manner, one can better compare the postoperative results.

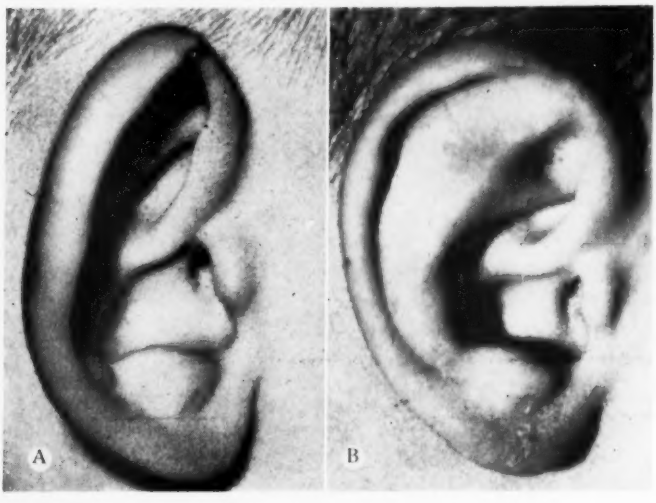

Fig. 7.—*A*, preoperative ear. This was operated upon employing my parallel cuts through the cartilage diagonal to the desired fold. *B*, postoperative. There is an irregularity of the antihelix which is not pleasing, owing to some of the cut strips of cartilage building up on the anterior surface.

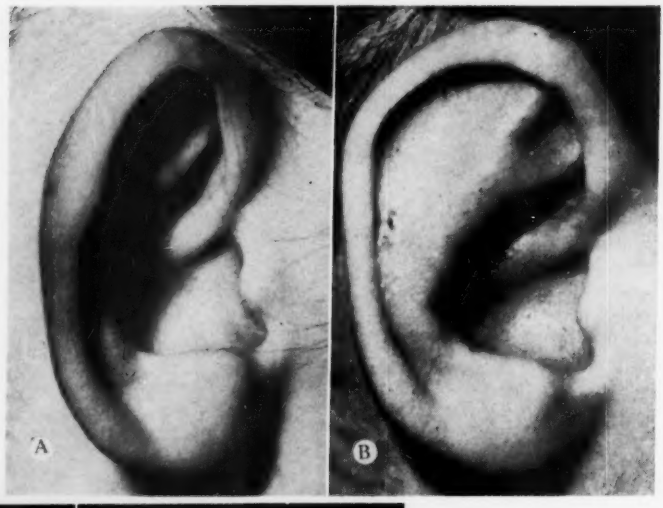

Fig. 8.—*A*, preoperative ear in which multiple, interdigitating cuts were used diagonal to the desired fold. (See Fig. 3). *B*, Postoperative. The postoperative result is better than in Figures 6 and 7, but occasionally irregularities are encountered using this method. This technique I employed for several years.

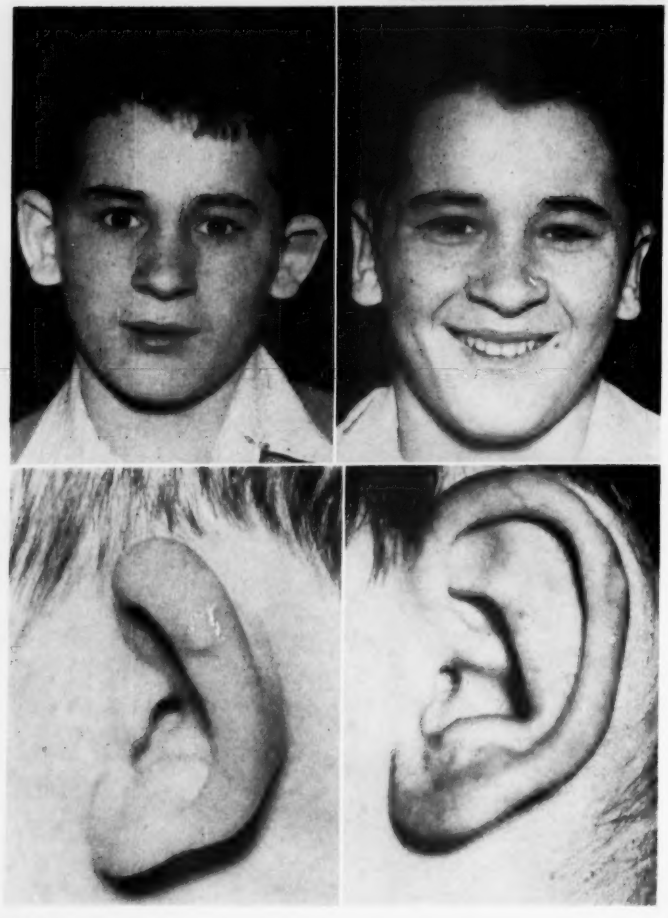

Fig. 9.—Case operated upon employing my newest technique of breaking the spring of the cartilage by creating "scales" of the bulk of the cartilage but not disturbing its anterior surface (Figs. 4 and 5*C*). The creation of an antihelix and superior crura without evidence of surgery has been accomplished.

from the front surface through the skin and cartilage, across the valley of the antihelix in back, then cut through the cartilage and skin to the front surface again. The sutures are then tied over petrolatum gauze rolls so as not to cut into the skin (Fig. 5*B*).

When the ear has been folded to its proper position, it will be observed that there will be a small excess of postauricular skin. This is excised so that the suture line falls in the normal, postauricular fold where it is inconspicuous. It is then closed with a continuous atraumatic suture. A very careful pressure dressing is next applied over this petrolatum gauze. This pressure dressing is most important so that no hematoma will develop. For such a purpose, one may use dental compound or cotton moistened with benzoin tincture or merely cotton waste. I find the benzoin tincture-cotton combination works very well, as we may pack the cotton where we wish to create pressure, and as it dries it forms a cast which helps to maintain the immobility of the ear while at the same time it prevents any blood or serum which may be present from decomposing and becoming foul. The dressing is left alone for one week, when the stitches are removed. In children, another protective dressing is applied until healing is complete.

Summary

A new technique is offered which will overcome the cartilaginous spring of an auricle, permitting the surgeon to bend a malformed ear to a more normal position without creating secondary undesirable deformities or irregularities.

330 Dartmouth St.

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Unusual Foreign Bodies in the Upper Respiratory Tract

Ten Cases of Leeches

CONSTANTIN A. FALIDAS, M.D., Lamia, Greece

The leech is, apparently, an exceptionally rare foreign body. In Greece some pertinent cases have been reported by Douvlaris,[1] by Polyzoidis,[2] and by Pessach.[3] In the present report we shall describe the largest series of cases in which leeches were found in the upper respiratory tract of humans. The large number of our cases may be explained by the special conditions of the water supply of the particular geographical area from which our patients came. (This is a low land in the center of Greece, to the north of Athens.)

Before we enter into the description of our cases, it might be expedient to review briefly the vital characteristics (biology) of the roundworms that are known under the name of leeches.

The leeches belong to the zoological phylum of Annelida (ringed worms), and more specifically to the order of Hirudinea. They have a flattened body, composed of many segments (rings), and their size varies from 5 to 350 mm. They have a strong muscular system consisting of three layers of muscular fibers. The external layer is transverse in the arrangement of its fibers; the middle one is oblique, and the innermost is longitudinal. Such an arrangement permits an undulant mode of movement. Locomotion is greatly helped by two suckers, one anterior and one posterior.

On their hosts, the leeches are attached by the anterior (oral) sucker. Their mouth lies in the middle of this anterior sucker, and it is equipped with a system of three toothful jaws. By moving these jaws in a lateral (retrogressive) manner, like saws, the leeches pierce a triangular opening in the skin or the mucous membranes of the host through which they suck blood, this being their principal food. The quantity of the sucked blood varies with the size of the leeches. The small ones suck 1 to 2 gm., that is, three times their body weight; those of a medium size suck 7 to 8 gm., that is, six times their weight, and the larger ones suck as much as 15 to 16 gm. When they have sucked an amount of blood sufficient for their needs, the leeches detach themselves from, or remain attached to, the host in an inert state. But the wound that has been caused on the host continues bleeding because of an enzyme, hirudin, which is secreted by the salivary glands of the parasite and which prevents the clotting of the blood.

The digestive system of the leeches consists of a simple tube that runs all through their body; it has two lateral furrows. The anus opens in the posterior sucker, through which also the surplus blood is excreted. The leeches are aquatic organisms that obtain their first nourishment from larvae of insects and from various small animals.

In the past, when bloodletting was widely practiced in medicine, the culture of leeches was a thriving business, mainly in Hungary, Turkey, and Russia. Since the middle of the 19th century, the medical use of the leeches has declined to the point that it has, practically, been abandoned today.

The leeches of medical importance are found in springs, brooks, and stagnant fresh water, where, from June to August, they deposit their eggs on aquatic plants (leeches are hermaphrodite); the young organisms

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are hatched six to eight weeks later. The growth of their body is slow. They live 15 to 20 years. They are not poisonous, nor are they intermediate hosts for other parasites of man.

As with the cases reported below, the leeches enter the human system with the drinking of water from an infested spring or brook. These are mainly collections of water to be found in the plains. The inhabitants of the nearby villages are aware of the fact that their springs contain leeches; for this reason they filter the water many times, passing it through a thick, folded gauze. As foreign bodies in man, leeches are to be found in the summer months. During this period the young animals are hatched, and their small size makes them difficult to discern; it is during the same season that the villagers stay in the fields for their work, using the water of these springs.

The entrance of a leech into the human system often is but a casual incident which timely medical intervention terminates without disagreeable consequences; however, sometimes it creates serious disturbance in the patient as well as in his environment, and it may lead to diagnostic problems, as will be seen in the cases below.

Case 1.—A man, 46 years of age, from Ombriaki, Domokou. He reported loss of bright red blood from his mouth, varying in quantity over a period of five days. He also complained of itching sensation in his larynx. General physical examination and chest plate were essentially negative. On Aug. 5, 1952, the patient came to our clinic.

Laryngoscopic examination revealed a small body of blackish shade, slightly mobile, about the anterior angle of the larynx. After anesthesia with tetracaine (Pontocaine), 2%, and by means of indirect laryngoscopy, we extracted the described foreign body. This was found to be a live leech. The patient recovered uneventfully.

Case 2.—A boy, 12 years of age from Stavros, Phthiotidos. He reported frequent episodes of epistaxis accompanied by loss of blood from the mouth, of a week's duration. Anterior packing of the nose and treatment with antihemorrhagic medication by a general practitioner failed to cure the patient, the hemorrhage recurring two to three times every 24 hours. The patient came to our clinic on Sept. 1, 1952.

Anterior examination of the nose did not reveal any hemorrhagic point. On examination of the oral cavity we observed in the pharynx a bloody coating extending upward behind the soft palate. Posterior rhinoscopy revealed a leech of considerable size attached to the roof of the rhinopharynx. With the aid of the proper forceps we succeeded in extracting the leech.

Case 3.—A 35-year-old woman from Stavros, Phthiotidos. She reported loss of blood from her mouth over a period of four days. Suspecting that she might have swallowed a leech, she came to our clinic on Aug. 1, 1955.

On examination of the oral cavity we discovered a leech moving slightly about the posterior wall toward the hypopharynx. With the proper instrument we extracted the worm.

Case 4.—A man, 55 years of age from Bardali, Domokou. He reported hemoptysis without fever or other disturbance, over a period of four days. A general practitioner treated him with bed rest and antihemorrhagic medication without result. A chest plate revealed nothing positive. The patient came to us on Aug. 13, 1955.

Indirect laryngoscopic examination revealed the presence of a small leech attached a short distance above the right vocal cord. After local anesthesia with tetracaine, 2%, we extracted the parasite easily. A slight laryngeal edema subsided within two days with the use of antibiotics.

Case 5.—A woman, 36 years of age, from Bardali, Domokou. She reported nasal hemorrhage of three days' duration. She suspected that her illness was due to a leech. She came to our clinic on Sept. 19, 1955.

Examination of the nose revealed nothing positive. On examination of the pharynx, we observed a large leech attached to the posterior wall of the rhinopharynx, behind the uvula. With the proper forceps we extracted the worm.

Case 6.—A man, 40 years of age, from Stavros, Phthiotidos. He reported loss of blood from his mouth without fever or other disturbance, over a period of four days. He came to us on Sept. 24, 1955. A chest plate revealed nothing positive.

Anterior and posterior examination of the nose, as well as laryngoscopic examination, was negative. In the course of the laryngoscopic examination we observed that the right tonsil was bleeding slightly. On closer examination of this tonsil we found a minute leech in the inside of one of the crypts, about the center of the tonsil. We extracted the leech, and the bleeding ceased.

Case 7.—A woman, 28 years of age, from Stavros, Phthiotidos. She reported hemorrhagic episodes from her nose and mouth over a period of three days. Like some of the previous cases, she suspected that the cause of the bleeding might be a leech. She came to us on Sept. 21, 1956.

On anterior examination of the nose we observed clotted blood in both nostrils. On examination of the oropharynx, we observed bloody coating on the posterior wall, extending upward behind the soft palate. Finally, on posterior examination of the nose we discovered a leech attached at some height near the roof of the pharynx. With the aid of the proper instrument we extracted the parasite.

CASE 8.—A woman, 80 years of age, from Bardali, Domokou. She complained of episodes of epistaxis over a period of eight days. She also complained of feeling very weak. She came to our clinic on Oct. 29, 1956.

On anterior rhinoscopic examination we observed a moving blackish foreign body about the posterior orifice of the left nasal cavity. Attempt to seize it with nasal forceps failed; subsequently, by means of posterior rhinoscopy and with a curved forceps, we seized and extracted the described foreign body, which proved to be a leech.

CASE 9.—A woman, 75 years of age, from Bardali, Domokou. She reported loss of blood from her mouth, accompanied by light cough, over a period of 11 days. She also remarked that her voice had become hoarse in the last 24 hours. Chest plate and general examination had revealed nothing positive. She came to us on Aug. 11, 1957.

On indirect laryngoscopic examination we observed a blackish foreign body attached to the right vocal cord, toward the posterior angle. After local anesthesia with tetracaine, 2%, by means of indirect laryngoscopy we proceeded to the extraction of the foreign body, which proved to be a leech. Antibiotics were administered, and the patient's voice (along with some laryngeal edema) cleared up within four days.

CASE 10.—A boy, 1½ years old, from Bardali, Domokou. Loss of blood from the infant's mouth and nose over a period of four days was reported by the frightened parents. The child was brought to our clinic on Aug. 26, 1957.

By applying pressure on the child's tongue, we were able to observe a very large leech attached firmly to the posterior wall of the pharynx. With the aid of the proper forceps we proceeded in the extraction of the leech.

Conclusions

The leech is a very rare foreign body that can cause anxiety and other serious disturbances in the human host; it may also give rise to diagnostic problems.

Early medical intervention may prevent a serious loss of blood and spare the patient much anxiety.

The construction of modern works for the proper water supply can deliver the population from the threat of the leeches.

Finally, appropriate education of the population, that should be warned not to drink water without prior filtration, may serve as a temporary preventive measure.

Summary

Ten cases of rare foreign bodies (leeches) of the upper respiratory tract have been reported in this paper. Three cases refer to the larynx, four to the rhinopharynx, two to the oropharynx, and one to the tonsils.

The biology of leeches is reviewed briefly.

Ainianon 6.

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Semeiology of Substernal Adenoma of the Thyroid

WILLIAM O. LODGE, M.D., Halifax, England

This is a study of the symptoms and signs of a condition found in about 1 in 50,000 of the community. It is of insidious onset and extended life history. It enters into the differential diagnosis of cancer of the lung but justifies a far better prognosis.

Embryologically, the thyroid gland is derived from the pharynx. In the course of numerous consultations and 325 biopsies and tracheal decompressions I have met with 7 cases in which goiters were not palpable in the neck, but were occult and intrathoracic. Of these one was inoperable. I operated on six successfully, and the patients survived. To operate regularly in the neck is essential if one is to perform major operations such as laryngectomy.

From the patients' point of view, the best person to perform such operations is whoever has the most intimate knowledge of the course of the recurrent laryngeal nerves, has the best judgment, and leaves the neatest scars. No true surgeon ever regrets removal of a tumor or cyst; sometimes a growth of a parathyroid or other pathological curiosity, potentially or actively harmful to the patient, comes to light.

Drs. Lindskog and Goldenberg [1] of Yale University, define a goiter as substernal if its lower border is shown radiologically to reach the level of the transverse process of the fourth dorsal vertebra. Mr. Hoffman [2] of Newcastle-upon-Tyne, describing 32 cases, accepts a goiter as intrathoracic if its greatest diameter is below the thoracic inlet. In all his cases the goiter descended as far as the aortic arch.

Until I read the text on bronchology of Drs. Soulas and Mounier-Kuhn [3] of Paris, I accepted the nebulous notion that early substernal adenoma produced no symptoms but dyspnea on exertion. Now I realize that to look only for respiratory symptoms is a mistake.

The trachea is of larger dimensions than is necessary for the tidal air; a proof is that it is possible to breathe through a tracheotomy tube of smaller dimensions than the trachea. Surely the value of a wide-bore trachea is to act as a reservoir of air. Complemental and supplemental air are most valuable in all states of unusual activity. Residual air is useful in swimming and diving. In that aquatic mammal the bottle-nosed seal, further provision for rebreathing has been acquired. In man, a reservoir of air is essential for speech and song.

It thus comes about that considerable deviation of the wide and flexible trachea to the right or left by a substernal adenoma is possible without any serious embarrassment of respiration. Compression is another story: pressure atrophy of the rings produces a scabbard trachea and causes dyspnea. It is in such cases that peroral endoscopy is dangerous. When the trachea is merely displaced laterally, endoscopy is not attended by much risk. In such cases bronchoscopy is a misnomer since it is the trachea that is being most critically examined.

Whenever the trachea is displaced the esophagus goes with it, but dysphagia is seldom an early symptom of substernal adenoma. A barium swallow is one of the simplest ways of demonstrating lateral deviation.

The recurrent laryngeal nerves are relatively proof against lateral displacement. A substernal adenoma on the right side is situated mainly below the level to which

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Otolaryngologist to the Hospitals of Halifax and Huddersfield.

the recurrent laryngeal nerve has been drawn down by the embryological descent of the subclavian artery. These nerves are, on the other hand, exceedingly vulnerable to inflammatory or malignant infiltration. The phrenic and sympathetic nerves show similar responses.

The innominate and carotid arteries with their elastic walls and contained blood, propelled by the rhythmic contractions of the left ventricle, are also able to overcome considerable mediastinal displacement.

The lymphatics usually reach the junction of the jugular and subclavian veins unimpeded by substernal adenomas.

Where, then, is the pressure first felt, and where must one look for the first symptoms and signs of retrosternal adenoma? That the answers are in the veins and in the manifestations of venous back pressure is borne in upon me by reconsideration of my own cases. I also find support for this view in contemporary literature. Dr. Burgess [4] of Manchester, noted hemoptysis as the presenting symptom in 3 out of 15 cases.

The trachea derives its blood supply from the inferior thyroid arteries. In the presence of substernal adenoma the inferior thyroid, innominate veins, and superior vena cava soon become congested, as evidenced by distention of the superficial veins of the neck and thorax. Eventually, as the tragedy is played out, a collateral circulation becomes established whereby the blood from the head and neck returns to the heart via the veins of the trunk, femoral veins, and inferior vena cava. At an early stage the distended veins on the face and neck turn noticeably blue in cold weather. In a case which I shall presently describe epistaxis was the presenting symptom. Sometimes patients in this predicament complain of nightmares.

To take a careful history and to make a methodical clinical examination is invaluable, particularly in excluding aneurysms and diseases of the hemopoietic system.

Former thyroidectomy scars were not noted in any of my cases; this experience would appear exceptional. Again, none of the adenomas became toxic or more than locally malignant, and that only in one advanced case. There were no metastases.

Radiologically, the diagnosis may be foreshadowed by skiagrams in the usual anteroposterior and lateral projections. A bronchogram may demonstrate the deviation. The upper lobe bronchus may be found to be obliterated and the middle lobe or lingular bronchus draped round the tumor. Tomograms or angiograms may be necessary to confirm the diagnosis.

According to Mr. Flavell [5] of the London Hospital, radioactive iodine isotopes are taken up by thyroid tissue and so may be used in the differential diagnosis of retrosternal adenoma.

A case which gave me confidence in bold surgery but which I do not propose to describe in detail was that of a woman in her 10th pregnancy, who complained that when one of the babies cried upstairs, whilst she was getting the older children ready for school down below, she was compelled to pause for breath half way up the stairs, and sometimes could go no further. Venous congestion of the head and neck and radiology lead to the diagnosis of substernal adenoma of the thyroid. A large aberrant adenoma was successfully removed at the sixth month of pregnancy. The patient's only comment at the conclusion of the treatment was that she could not understand what all the fuss was about.

To any responsible and scientific medical man it is evident that there is only one way to reduce such risks and that is by early diagnosis.

I have only one case which I wish to present *in extenso*, but before doing so it may be of interest to readers to compare attitudes toward what was there the presenting symptom, epitaxis.

Epistaxis may be due to a thousand and one different causes ranging from the onset of measles to the termination of typhus. There must be few persons living who have not experienced it at one time or another. In Osler's disease the prognosis is bad. Injection of iodized oil into the maxillary

antra may reveal early carcinoma. Sometimes splenic and lymphatic enlargements point to leukemia. In hypertensive cases a week's in-patient treatment will usually suffice, but when the epistaxis is intractably recurrent ligation of the external carotid or of the internal maxillary artery, transantrally, will ensure good nights' rest for the patient and doctor; the hemorrhage may recur but will no longer be catastrophic. Now, it seems, the possibility of an occult substernal adenoma must also be borne in mind, in routine investigation.

Although the Mounier-Kuhn syndrome is a Procrustean bed for the clinical entity about to be described, it will promote a ruthless presentation of the case.

An eminent colleague, aged 63, had always enjoyed good health except for right apical pneumonia about 10 years ago, from which he made a good recovery. Once in the past—how long ago he could not say—there had been an attack of dyspnea during a holiday in the Lake District; then—looking back—something seemed to change position so that breathing became free again and he was as active as any of his contemporaries.

Dilated capillaries on the face and neck, which turned blue in cold weather, resembled lupus erythematosus but there was no suggestion of ulceration, at least exteriorly; within the nose a vascularized area on the nasal septum was evidently responsible for the symptom which caused me to admit him to hospital for investigation, namely, epistaxis, which had been recurrent for a few weeks but receded from the picture after prescriptions for vitamins K and C, internally, and scarlet red ointment locally.

Apart from slight recent loss of energy he had always felt well. If he had an occasional dry cough, it certainly never had any barking or reverberating tone or abnormal resonance, nor did it ever take on a muffled or muted quality when he had a cold in the head. The voice was quite clear, and when he declaimed verse, of which he has an extensive repertoire—being, as he justly states, a physician and metaphysician—he never has any difficulty in reaching a caesura.

Stridor was as little inspiratory as expiratory, for it simply did not exist. There was no tracheal tugging. Nocturnal asthmatic attacks were absent; there had never been any serious attack of dyspnea.

When I put it to him that perhaps sometimes when his vigilance had been relaxed there had been a purring sound, he repudiated any such suggestion; furthermore, there was no question either of improvement of respiration by change of position or effort of will. He did not suffer from nightmares. There was no diphonic or other alteration of the voice.

Dr. John Follows, consultant physician, whom I called in, found diminished movement, resonance and air entry over the right upper lobe, and as the investigations proceeded, made the final diagnosis. The movements of the vocal folds were normal. Clinical examination from head to foot revealed no other abnormality.

Dr. J. W. Weston, pathologist, reported the following: hemoglobin 90%; red cells normal in appearance; polymorphonuclears 56%; eosinophils 2%; basophils 1%; large lymphocytes 29%; small lymphocytes, none; monocytes 12%; platelets 175,000 per cubic millimeter; W. R. and Kahn negative; bleeding time 4 minutes; clotting time 6 minutes; prothrombin content 100%.

On x-ray examination of the chest an opacity was seen to occupy the superior mediastinum and right apical region. There was widening and unfolding of the aortic arch.

An electrocardiogram revealed no abnormality. The blood pressure was 120/80.

On peroral endoscopy the trachea and esophagus were found to be deviated to the left; one got the impression of atelectasis of the right upper lobe and of participation of the right middle lobe bronchus in the embrace of a tumor. Under the

Fig. 1.—Tomogram showing a partially calcified substernal adenoma of the thyroid embraced by the trachea and middle lobe bronchus. The right upper lobe is in a state of partial atelectasis. Yet there is no respiratory distress; though the trachea is deviated, it remains wide in caliber. Whether epistaxis is to be accepted as the presenting symptom is for the reader to judge; the patient—a physician—is convinced that this was so.

Fig. 2.—Tomogram taken three weeks after removal of the substernal adenoma shown in Figure 2. The trachea is back in its proper position, and the right upper lobe of the lung has expanded again.

anesthetic, by the deepest possible downward pressure from the suprasternal notch, one could imagine that one felt the upper surface of a tumor.

During this examination the dangers of peroral endoscopy in tracheal compressive states were borne in mind.

Further radiology was clearly indicated. In due course Dr. R. I. Lewis reported that tomographs "demonstrate a large calcifying soft-tissue mass at the level of the thoracic inlet; this mass compresses the right lateral wall of the trachea and the latter is deviated to the left. A similar picture may be presented by a calcifying adenoma of the thyroid."

On Jan. 2d, 1958, under general anesthesia by Dr. F. W. Oliver, excision of the calcified adenoma of the thyroid was carried out, through a collar incision of the usual type. The tumor was 10 cm. in diameter and occupied the superior mediastinum on the right side. It was not feasible at the outset to ligate the inferior thyroid artery, but this was done when the mass was delivered from the thorax up into the root of the neck. The superior thyroid vessels were also ligated on the right side. The upper pole of the partially encapsulated adenoma was surrounded by a plexus of dilated veins. These were encircled by absorbable surgical sutures and drawn together. Inferior thyroid veins connecting the tumor to its bed were ligated, thermocoagulated, or treated with fibrin foam, according to their size and situation, with particular care for the dome of the pleura, which was not opened, and for the recurrent laryngeal nerve. The patient's eloquence and success as a hypnotist, and his consequent dependence upon a well-modulated voice, were borne in mind. The wound was closed with a separate small incision for drainage; it was fortunate that ample provision was made, for a sanious discharge continued for nearly 14 days, although the dead space had been closed as far as possible by capitonnage; that is to say, by successive purse-string sutures.

Dr. R. A. Garson found no malignant changes in the partially calcified thyroadenoma.

There was no return of the epistaxis and the dilated vessels on the face became much less evident.

I advised the patient to take a long holiday, but he pleaded to be allowed to return to work and did so within a few days of leaving hospital, taking up the deputy control of a vast establishment, in the absence of his chief. The scar quickly became almost invisible.

What would have been the outcome if the patient had been less courageous? The plight of a man aged 80 who had had a substernal goiter for 40 years and had been treated conservatively is recalled. The tumor eventually displayed evidence of local malignancy, but its main effects were of venous obstruction and pressure atrophy; the venous return from the head and neck were completely obstructed. Large veins coursed over the chest and abdomen, returning blood to the heart via the femoral veins and inferior vena cava.

Summary

The manifestations of substernal adenoma of the thyroid in the last of 6 cases, encountered in the course of 325 operations on the gland, have been critically reviewed in the light of Soulas and Mounier-Kuhn's important recent work.

That the presenting symptoms and signs should have been due to venous back-pressure, rather than to the admittedly very real element of respiratory obstruction, calls—in my submission—for the recognition of more than one syndrome.

It is certainly unscientific to stumble upon retrosternal adenoma of the thyroid by chance, but early diagnosis is quite a problem.

Due investigation of epistaxis or hemoptysis may bring to light this occult but innocent tumor, which will make life

miserable, eventually, if neglected, but may be dealt with—by well-timed surgical intervention—with minimal mortality.

Green Leas, Savile Park.

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Rhinophyma

FRANK E. FISHOF, M.D., Wilkes-Barre, Pa.

A case of rhinophyma is reported because it presented two surgical problems involving the nose; namely, removal of the hyperplastic growth and the correction of a drooped nasal tip. This growth[1,2] is a distinct entity in that pathologically it is characterized by hyperplasia of the sebaceous glands, widening of the follicles, keratinization and formation of comedones, thickening of the cutis, and telangiectasia. It is preceded by a rosacea type of dermatitis manifested by transient episodes of erythema and vasodilatation. In the course of its evolution, papules and pustules begin to appear. This may take place several months or years after its original onset. With repeated recurrences, the condition becomes more lasting and drastic. The rosacea most frequently involves the flushed regions of the face, such as the lower half of the nose, paranasal regions, and chin. It may also extend to the appendages of the eyes, conjunctiva, and cornea. Occasionally the external ear may be affected along with the nose, in which case it is called otorhinophyma.[4] When it is limited to the chin, it is termed mentophyma.[5] When the nose is involved, it is called rhinophyma.

Grossly most rhinophymas are of the smooth hypertrophic bulbous type seeped with sebaceous secretions and coursed by telangiectatic blood vessels. It has a dusky red or purple color. It is the kind with which the patient is not too much concerned, especially the less fastidious sex. He is willing to put up with it. The other, and fortunately the less frequent type, presents a multilobulated tumorous mass marred with fissures and occasional nodular proliferations. It is noninflammatory, of fibrotic consistency (almost scirrhous), and mottled with black and white comedones, especially at the lower third of the nose. It too presents numerous dilated vessels. Both types of rhinophyma are considered the end-stage of rosacea. In fact, Beerman[3] states: "It may be the sole evidence of the existence of this process." Most patients, who have unfortunately harbored the disease to the stage of rhinophyma, have developed in the process of evolution of this condition an inferiority complex associated with the nasal deformity. It may aptly be termed the rhinophyma-nose complex.

Because of its peculiar characteristic appearance, this grotesque mass has been dubbed whiskey[6] or brandy nose, growing nose, rumblossom nose, double nose, *Kartoffel* (potato) nose. From the structural aspect,[7] the condition is identified as acne hyperplastica, cystadenofibroma, fibroma molluscum of the nose. It may also be complicated by a neoplastic growth such as basal-cell carcinoma.[18]

The etiology of rosacea per se is still controversial. Different authorities attribute different probable causes depending on their experience with the condition. Almost all agree that clinically it starts out as a facial disorder characterized by transient episodes of erythema and vasodilatation. It is never explosive. It generally manifests itself in the third or fourth decade although it may appear earlier or later in life. It may mirror internal medical disturbances with expressions in gastrointestinal and neurovascular disorders. This condition occurs mostly in patients who present vasomotor instability, emotional and nervous states, disorders of the fifth nerve pertaining to mediation of stimuli to the vaso-

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Chief, Eye-Ear-Nose-Throat Section of Surgical Service, Veterans Administration Hospital.

dilator and vasoconstrictor centers in the brain.

It is felt that the threshold to afferent impulses along the path of this complex nerve is lowered to such a level that toxic products from imperfect digestion, exposure to extreme changes in climatic environment, and breakdown in cellular respiration by deficiency in riboflavin are contributory to the precipitation or aggravation of the disease. Stimulating beverages, such as coffee, tea, and alcohol, and an abundant use of condiments also play a contributory part in that condition. Carbohydrates taken in large quantities may produce an overloading of the dilated blood vessels with sugar. This is especially true when there is a disturbance in carbohydrate metabolism. Under these circumstances, the sebum-producing glands are more active, especially in cases that have the "seborrheic habitus." The skin becomes soggy with oily secretions and vulnerable to infection. Demodex folliculorum is an example of one of the mites that occasionally invade the site of the rosacea complex.

Treatment

The therapy of rhinophyma-rosacea complex consists of medical and surgical measures. In the prerhinophyma stage, it belongs in the domain of the dermatologist. There are some contributory factors that may be carried over from the early stages of this disease to the terminal period of the rosacea complex (rhinophyma); or these causal backgrounds may become aggravated or precipitated in the rhinophyma stage. Superimposed infections have favorably responded to antimicrobial agents, such as sulfonamides, chlortetracycline, oxytetracycline, quinacrine (mepacrine) hydrochloride, and dihydrostreptomycin. To avoid promiscuous use of antibiotics, sensitivity tests of the invading organism should be done. During the climacteric period, hormones have been used to minimize vasomotor instability. Corticotropin (ACTH)[8] and progesterone provided some improvement. Androgenic hormones[17] should be avoided. They may provoke sebaceous gland hypertrophy. Additional measures, such as avoidance of exposure to extreme climatic changes, refraining from use of sweets, spicy foods, and stimulants, such as coffee, tea, and alcoholic beverages, are in order. Avitaminosis should be avoided, especially in regard to riboflavin (B_{12}). This vitamin combines with various body proteins to form Warburg's yellow respiratory enzyme, which takes part in tissue respiration. It is especially important in avascular tissue, such as cornea and lens. Anacidity is controlled by the use of hydrochloride capsules. The treatment of the emotional trauma, sense of insecurity, and inferiority complex that has been built up in relation to the rosacea-rhinophyma complex should be relegated to the psychotherapist.

The direct treatment of rhinophmya per se varies considerably. Cryotherapy in the form of solid carbon dioxide or liquid nitrogen has been used by some dermatologists. A sclerotic solution, such as trichloroacetic acid,[9] has been used to inject the growth. Shrinkage of the mass by scarring the tissue[10-12,19] with monopolar electrodes such as the Hyfrecator or diathermy has also been applied. Some have resorted to the use of roentgen radiation and radon implantation to reduce the tumerous growth. These modes of approach are not very satisfactory because they do not remove the pathological condition. They merely decrease its size. In the case of the multilobulated type of rhinophyma in which deep scarring is present and which accounts for the furrows and lobulation, the abnormal configuration of the nose still remains even though its size has been reduced. The case reported by Villafuerte[13] speaks strongly against the use of x-ray therapy. The deformity it produced was worse than the original condition. It also impaired the physiological function of the nose.

Surgery that aims at eradication of the pathological condition is the treatment of choice. This may be accomplished by use

of either cold scalpel [14,16,19,20] or "electric knife." [15] In the latter modality, the electrode may be a sharp one or a loop. When the scalpel is used, the growth may be removed by paring in gradual stages; or the lesion is circumscribed by a deep incision extending through the full cutaneous depths and then the growth is decorticated en masse. The raw surface may either be allowed to epithelize spontaneously or be covered with a full-thickness or split-thickness free skin graft. The monopolar electrode may be used to spark bleeding points.

The electric knife or loop is used to shave the growth from its underlying surface. The partly desiccated surface may then be allowed to undergo primary epithelization or be skin-grafted as mentioned above. This approach I do not favor. I feel that one has less control in avoiding encroachment on the osseous cartilaginous dome. The superficial blood supply is unduly interfered with by application of the cautery. With the use of the cold knife, cautery is applied only where and when bleeding occurs that cannot be controlled with pressure.

Report of Case

A white man, age 65, automobile mechanic by trade, was admitted to the E. E. N. T. Section on Aug. 8, 1957, for treatment of nasal obstruction and a growth on the exterior of the nose. History revealed that at the age of 15, in 1907, he developed some pimples on the face, neck, and to some extent on the chin. He had no medical care for the condition. In 1919 at age 27, when he was discharged from the Army, the rash had completely disappeared. The nose had a normal appearance. A submucous resection of the nose was performed in 1922 (age 30). About 10-12 years prior to admission, he developed a sense of irritation inside the nose and pustular pimples appeared on the exterior. These eruptions receded and reappeared at frequent intervals. During the cold season, the condition was more pronounced. The skin would get irritated and red; nasal secretions increased, and he found himself constantly wiping his nose.

The nasal bulb gradually increased in size. The skin lost its natural sheen and resilience. It became purplish-red and somewhat hard to touch. Numerous fissures and irregular nodules disfigured the natural contour. It became somewhat puckered. For the past two years, the nose had not undergone any change in size, shape, or color. Within two years prior to that period, however, alterations had become so rapid and pronounced that he became extremely conscious of the condition. The mass increased in size to the extent that it tipped the bulb labialward, producing partial obstruction to nasal ventilation. The shape became so grotesque in appearance that he developed a "nose complex." He stated: "I never felt at ease when meeting new people. I was always conscious that they were looking at my nose rather than at me as an individual. I just did not feel I could do the job right."

He complained of gastrointestinal disturbances characterized by a tendency to belching, constipation, and a bloated feeling. Glutamic acid hydrochloride capsules seemed to aleviate the condition. He sweated considerably on the forehead and back of the neck. He denied excessive use of alcoholic

Fig. 1.—Rhinophyma of several years' duration.

beverages. He consumed about two to three cups of coffee a day and smoked about a pack of cigarettes a day.

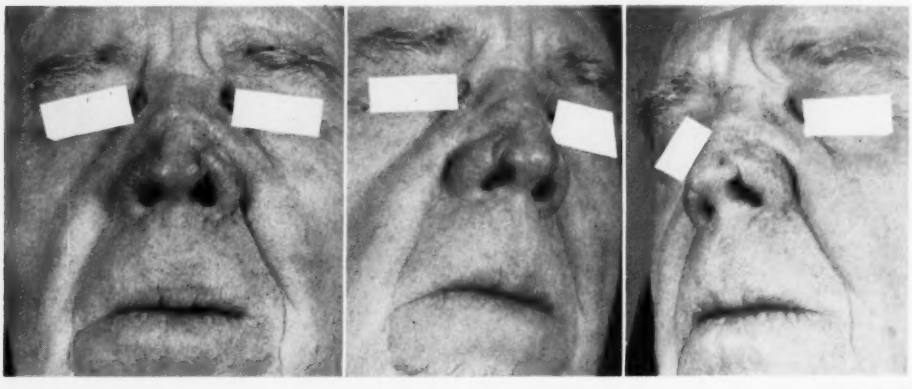
Physical Findings.—General physical examination did not display any significant abnormalities. Roentgen and laboratory reports were within normal range. The nose (Fig. 1) displayed a large, benign, hyperplastic, lobulated, fissured, grotesque mass extending from the lower border of the nasal bones to the margin of the rim of the nose and laterally involving the lower and upper nasal alae. There were numerous comedones and depressions of old keratinized follicles. The skin was dark red interspersed with spots of purplish hue. The lower right ala at the level of its upper and middle third (junction of convex and concave surface) was depressed downward and inward, almost obliterating the vestibule. The skin covering the convex portion of this ala was of normal color and consistency. The external surface of the left ala had the same characteristics as the main growth. The internal surface was normal. The nasal mucosa appeared somewhat dry and was covered with flakes of crusts. The entire septum was gone with the exception of a small cartilaginous and membranous portion and the columella.

Operation.—The dorsum of the nose and surrounding tissues were aseptically prepared, using Phisoderm* and benzalkonium (Zephiran) solution (1:10,000). The interior of the nose was cleansed with peroxide and sprayed with benzalkonium solution to rid it of thick mucoid secretions and crusts. Attention was first directed at correcting the drooped tip of the nose. A solution of procaine (Novocain) 1%, with 8 drops of epinephrine (Adrenalin) to the ounce and hyaluronidase 75 units to the ounce, was used to anesthetize the columella and a very small residual portion of septal cartilage. A vertical incision was made in the posterior part of the columella to construct a pocket for reception of a strut to raise and support the pendulous tip of the nose. Septal bone procured from "the bank" was used to shape the desired pillar. The lower part of this strut, which terminated in a sharp angle, was fitted into a prepared recess anterior to the spine of the maxilla. The proximal part, which was considerably wider, was directed toward the tip of the nose and adjusted to provide the desired cosmetic effect in the columella-labial angle. The implant was anchored by placing 000 silk sutures, one above and one below the center of the vertical axis of the implant. The posterior edges of the previously incised columella were approximated with three interrupted 0000 silk sutures. The latter maneuver was to prevent posterior displacement of the implanted bone.

The benign growth involving the lower two-thirds of the dorsum of the nose was resolved in the following manner: The above-mentioned anesthetic solution was used to anesthetize the outer limits of the deep surface of the rhinophyma. With use of a No. 15 Bard-Parker blade, a superficial incision was started at the upper margin of the columella about 3 to 4 mm. below the growth. The incision was extended lateralward to the right along the rim of the upper third of the lower lateral ala cartilage to the level where the deep dent in the soft tissue was present (junction of upper and middle two-thirds). The incision was then extended upward along the dent that followed along the nasofacial sulcus to the lower border of the nasal bone. The same procedure was followed to the left except that the incision at the anterior margin of the ala was carried down to the level of the middle with the lower third. The lower lateral borders of the growth were thus outlined. A No. 10 blade was then used to extend the incision deeper. The growth was gradually

* Detergent containing sodium octylphenoxyethyl ether sulfonate.

Fig. 2.—Rhinophyma 10 months after operation.



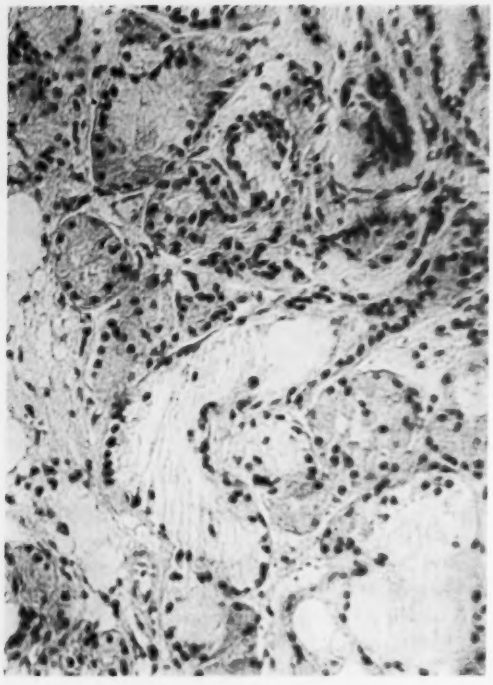
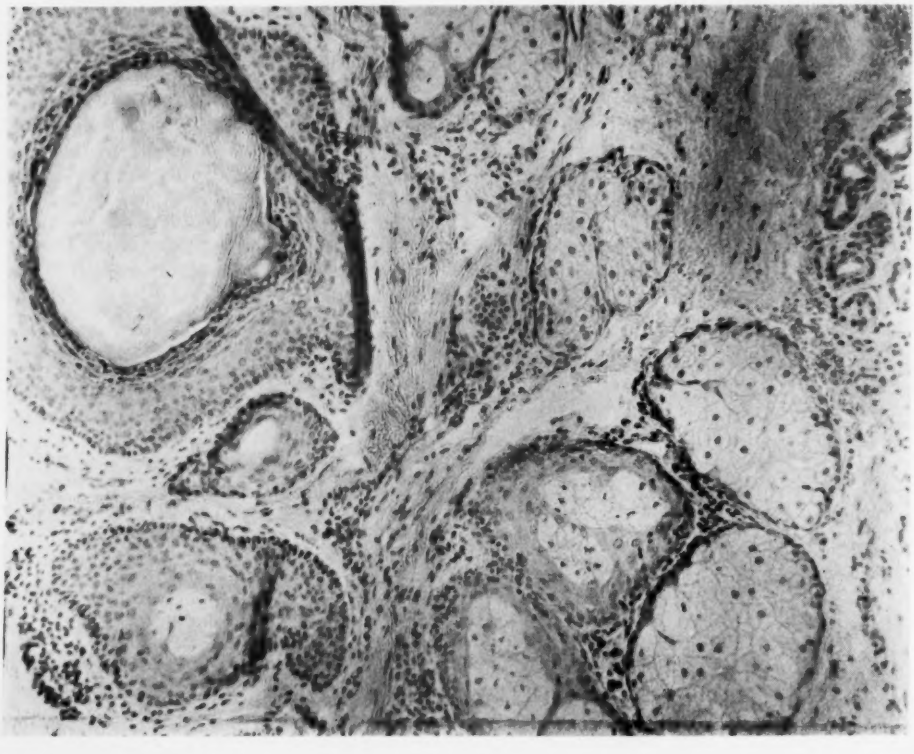


Fig. 3.—Low-power photomicrograph of decorticated rhinophyma. Note sebaceous glands filled with inspissated material and sweat glands filled with keratin.

Fig. 4.—High-power photomicrograph of decorticated rhinophyma. Note clogged follicles, keratin-plugged sweat glands, sebaceous glands filled with inspissated material, and hyperplasia of connective tissue.



raised from its undersurface as decortication was progressing. The knife was held slightly angulated with the sharp edge facing upward to avoid cutting into the cartilaginous vault. After nearly two-thirds of the growth had been undermined, the knife was then directed toward the upper limits of the growth near the distal margin of the nasal bones. Here, too, the initial incision was superficial and slightly above the limits of the growth. This was followed by a deeper incision. The knife was held slightly angulated with sharp edge facing downward. Decortication of the growth was thus completed. The mass was removed in toto. Warm saline solution dressing was applied for a few minutes to control slight oozing. The raw surface was covered with a layer of absorbable gelatin sponge (Gelfoam) that was dipped in a solution of penicillin, 300,000 units to the cc. It was topped by a layer of Telfa and gauze. The entire dressing was kept in place by three strips of adhesive and a figure-of-eight bandage. On the seventh postoperative day, the dressing was changed. Peroxide and benzalkonium solution (1:10,000) was used to loosen the absorbable gelatin sponge from its underlying surface. Care was taken not to disrupt the neovascularization and epithelization. The nose was redressed with a layer of nitrofurantoin (Furadantin) gauze followed with a thin layer of sterile gauze and three strips of adhesive.

The patient resumed work within two weeks after the operation.

Summary

Rosacea complex is a circulatory disorder characterized by transient erythema and vasodilatation of the flushed surface of the face. It is never explosive.

The condition is aggravated by (*a*) vasomotor instability; (*b*) gastrointestinal dyscrasia (anacidity, etc.); (*c*) extreme climatic changes; (*d*) climacteric changes; (*e*) abuse of stimulants, such as coffee, tea, alcoholic beverages, and spicy foods; (*f*) excessive use of carbohydrates.

Rhinophyma-rosacea inferiority complex is acquired during the evolution of the condition.

Treatment has been discussed from the medical and surgical aspects. The former aims to abhort the rosacea complex. Surgery tends to eradicate the growth and to restore normal cosmetic and physiological function of the nose.

A case of rhinophyma of the lobulated type complicated by a drooped nasal tip caused by previous resection of the septum is presented. The evolution of the condition has been traced. The development of the rhinophyma-rosacea inferiority complex was detailed. The nasal deformity was corrected by implantation of bone in the columella region procured from "the bank." The growth was removed with a scalpel. A superficial incision was first made to circumscribe the growth. A deep incision extending through the full cutaneous depths was then made to complete decortication en masse. The surface was allowed to epithelize spontaneously. The patient resumed work within two weeks.

The Chief Librarian, Miss Louise Geroulo, and the Medical Librarian, Miss Betsy Stathakis, assisted in the preparation of this article.

Veterans Administration Hospital.

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Rupture of the Common Carotid Artery Following Radical Neck Surgery in Radiated Cases

JULIUS W. McCALL, M.D.; C. W. WHITAKER, M.D., and E. L. HENDERSHOT, M.D., Cleveland

It has long been known that neck surgery following radiation therapy for primary and metastatic carcinomas is fraught with complications. These complications include necrosis of the skin flaps, oral and pharyngeal fistulas, and necrosis and rupture of the common carotid artery. Since the carotid artery derives 80% of its blood supply from its adventitia, and the adventitia is removed in the course of radical neck dissection, the artery is dependent upon the skin flaps for its future nourishment. Irradiated tissues with their decreased vascularity do not heal well after surgery, and no other region is more in need of adequate vascularity than the tissues of the neck. Failure of the flaps to heal predisposes to oral and pharyngeal fistulas and necrosis and rupture of the common carotid artery. While flap necrosis and fistulas can be treated with deliberation, rupture of the common carotid artery is a dire emergency demanding immediate attention.

Until recently, mortality rates of common carotid artery ligation varied from 4% to 60%. In the 19th century, common carotid artery ligation was a popular procedure employed for hemorrhage, tumors, aneurysms, epilepsy, trigeminal neuralgia, psychosis, headache, and many other conditions. However, Wyeth [1] in 1879 reported an overall mortality rate of 41%. De Fourmestraux [2] divided the ligation procedures into four groups and noticed a wide variation in mortality figures. In ligation for hemorrhage, the mortality rate was 54%; for tumors it was 46%; for aneurysms, 13.5%, and for pulsating exophthalmos, 7%. The relatively low mortality rate for ligation for aneurysms has been confirmed by Matas [3] (4%), Dandy [4] (5%), and Schorstein [5] (13.3%). Gurdjian et al.[6] report a 5% mortality rate in 63 ligations, 44 of which were for aneurysm. In the treatment of neck tumors, Watson and Silverstone [7] in 1939 reported 60% cerebral complications with 55% mortality rate, while Martin et al.[8] in 1951 reported a 40%-50% mortality rate.

Cerebral complications are caused by poor collateral circulation in the circle of Willis, shock or hypotension at the time of ligation, reflex cerebral vasoconstriction, ascending carotid artery thrombosis, and the general condition of the patient. In elective ligations, several tests may be used to determine the prognosis. The "Matas test" is mere digital pressure over the carotid artery with the overlying structures intact. Dandy [10] exposed the artery under local anesthesia and totally occluded it for a short time. A refinement to Dandy's procedure is to run an electroencephalogram concomitantly to observe changes in the brain waves. Dandy [10] in order to promote collateral circulation, partially occluded the artery for several days and then ligated it. The mortality rate has been lowered from 50% to 4.5% in this manner. To reduce or eliminate reflex cerebral vasoconstriction, one may interrupt the cervical sympathetic chain. Transection of the artery after doubly ligating it may further interrupt the sympathetic fibers.

When the common carotid artery ruptures, shock ensues rapidly. Ligation or repair of the defect must be performed immediately, with simultaneous blood transfusion. Conley [11] emphasizes the need for taking pres-

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From the Department of Otolaryngology, St. Luke's Hospital.

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sures in the internal carotid artery. If the pressure drops significantly (60%-70%), he recommends anastomosis of the internal carotid artery to the ipsilateral external carotid artery. However, the operating conditions, both operating place and the urgency with which the artery is ligated, frequently preclude the possibility of performing these worth-while procedures. Indeed, it is a tribute to excellent nursing care and alert house officers that these ruptures are detected before the patient becomes exsanguinated.

Report of Cases

The following cases are presented:

CASE 1.—A 55-year-old white man had a mass in the neck biopsied in April, 1956, which was diagnosed as metastatic, poorly differentiated squamous-cell carcinoma. ENT consultation revealed a matchhead-sized primary site on the posterior pharyngeal wall, which was surgically inaccessible. He received 45 x-ray treatments, which arrested the primary lesion. However, in May, 1957, a left radical neck dissection was performed for metastases. The flap beneath the angle of the jaw did not heal, and a pharyngeal fistula developed. In June a delayed pedicle flap was raised and on July 20 while he was being prepared for transplant of the flap, the common carotid artery ruptured. Repair of the defect was unsuccessful, and ligation was performed. Several days later, the flap was transplanted. No paralysis or paresis developed, and the patient made an uneventful recovery.

Fig. 1 (Case 1).—A pedicle graft based at the shoulder. Area from which the graft was taken covered with a split-thickness graft.

CASE 2.—A 61-year-old white man was operated on in October, 1956, for carcinoma of the pharynx. Following this he had 30 x-ray treatments. In April, 1957, he developed dysphagia and he subsequently lost 15 lb. Physical examination revealed a 5-cm.-diameter ulcer on the lateral pharyngeal wall. There were erythema and induration of the left side of the neck, but no nodes were palpable. On May 5, a left radical neck dissection was performed. On June 5, a right radical neck dissection, with excision of the pharyngeal lesion and application of a full-thickness pedicle graft to the pharyngeal defect, was performed. This defect was not completely closed, and a fistula developed. Eleven days postoperatively, he hemorrhaged on the ward and was taken to surgery where a 0.5-cm. defect in the common carotid artery was found. It was ligated and a split-thickness graft was placed over it for protection. Postoperatively, neurological examination revealed no evidence of paresis. On July 5, the oral fistula was closed with a pedicle graft. The patient died Oct. 21, 1957. Autopsy revealed no gross pathology in the brain.

CASE 3.—A 58-year-old white man was treated by x-ray for a poorly differentiated squamous-cell carcinoma of the tongue at the junction of the right anterior pillar of the tonsil in May, 1956. In December, 1956, biopsy of the soft palate revealed extension of the carcinoma, and in January, 1957, 12 radon seeds were implanted into the lesion. Subsequently the patient developed severe pain in his jaw, and he was unable to open his mouth. On May 5, he underwent a right radical neck dissection, partial mandibulectomy, and excision of the original lesion. The flaps did not heal, and he developed a fistula. On June 19, a pedicle flap was raised, and six days later the flap was tubed. The next day he hemorrhaged profusely, and a defect was found in the right common carotid artery. The artery was ligated after unsuccessful repair. No neurological sequelae developed. However, the preceding grafts have done poorly, and he has recently developed local recurrences. At present his condition is poor.

CASE 4.—A 51-year-old white man was treated by x-ray for an early intrinsic laryngeal carcinoma in the summer of 1956. The lesion did not regress, and on June 25, 1957, a right radical neck dissection with laryngectomy was performed. His postoperative course was benign, and progressive diets were begun on the eighth postoperative day. He

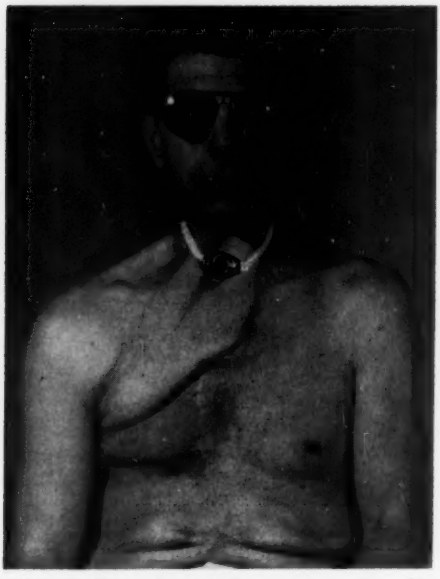

Fig. 2 (Case 4).—Utilization of a chest flap. Note the preservation of the nipple. The circular defect in the flap has been used to create a new tracheal stoma. Split-thickness skin graft covers the chest defect.

was readmitted July 16, 1957, with inability to swallow and with a fistula laterally in the supraclavicular area. Intubation was unsuccessful, the hypopharynx having developed severe edema, and after five days a gastrostomy was performed. On Aug. 12, 1957, the flaps sloughed, leaving a portion of the carotid artery exposed. Two days later he hemorrhaged profusely, and the common carotid artery was ligated. There was no postoperative neurological deficit. On Aug. 17, the necrotic tissue was excised, and a pedicle chest flap was placed over the wound. Healing has been retarded owing to his generally debilitated condition.

Comment

In analyzing these cases, five common features are prominent: (1) The patients had had previous radiation therapy to the neck; (2) the flaps did not heal; (3) fistulas developed; (4) the common carotid artery ruptured; (5) no neurological sequelae developed from ligation. It is fortunate that we can salvage these cases by ligating the common carotid artery, but the necessary plastic procedures and morbidity greatly increase the length and cost of the hospital stay, and cause considerable mental trauma.

Our treatment in these radiated cases is being directed toward prevention of complications. To nourish the common carotid artery, Grady, Robinson et al.,[12] divide the levator scapulae-muscle inferiorly, bring it across the carotid bulb, and attach it to the strap muscles anteriorly. Conley[11] states that if flap necrosis occurs, a flap should be advanced immediately. Degeneration of the flap with fistula formation accelerates carotid-artery rupture. We favor raising a flap at the time of neck dissection, and, if necessary, sacrificing radiated skin. If the skin is not sacrificed, the flap is sutured back into place and kept in reserve. This serves two purposes: (1) If the radiated skin should be sacrificed, the flap can be advanced immediately, and (2) if the skin is not sacrificed and subsequently necroses, the flap has had additional capillary stimulation.

Choice of flap site is important. It should not include radiated skin; nor should it include skin where there is likelihood of recurrence of tumor. Chest flaps based at the shoulder are excellent. These were used successfully in two patients (Cases 1 and 4). Zovickian[13] utilizes large shoulder flaps based superiorly at the mastoid and occipital regions to provide a permanent blood-carrying pedicle for the repair.

Finally, a word should be said about the causative agent of this morbidity-radiation therapy. Castigliano and Rominger[14] reported that in neck dissections following up to 5,000 r of radiation, the average healing time is prolonged 59%, but made no mention of flap necrosis or fistula formation. Although there are still many radiation enthusiasts, it is becoming increasingly apparent that radiation leaves much to be desired in the treatment of head and neck cancer. Not only is it ineffective in treating regional metastasis, but also a high percentage of primary lesions progress. Radiation ulcers at the primary site are common and can cause nearly as much distress as the cancer.

The surgical attack on head and neck cancer is becoming bolder. The increasing utilization of pedicle flaps for oral and

pharyngeal reconstruction has made possible adequate resection of primary lesions which were once considered inoperable. And with more extensive removal of the primary lesions, the cure rates are improving. There have been cases in which radiation therapy was chosen over surgery because the physician or patient considered radical surgery too disfiguring. We agree that there is some disfigurement in radical head and neck surgery but we also believe that there is no place for modesty in cancer therapy.

Summary

The hazards of radical neck surgery following radiation therapy are discussed with emphasis on carotid artery complications. Four cases of common carotid artery rupture and ligation with no subsequent neurological sequelae are presented. The authors believe that in radiated cases a pedicle flap should be raised at the time of dissection, and, if necessary, the radiated skin should be sacrificed with the specimen.

Conclusions

1. Rupture of the common carotid artery following radical neck surgery in radiated patients is not unusual.

2. Common carotid artery ligation does not carry with it the previously supposed mortality and morbidity.

3. Adequate surgery should be the treatment of choice for accessible lesions in the head and neck, with radiation reserved for surgically inaccessible lesions and as palliative treatment.

4. The more frequent use of pedicle flaps in the event of skin necrosis following surgery in radiated necks can reduce morbidity.

C. Kiehn, M.D., granted us permission to include two patients (Cases 2 and 3) in our series.

2060 E. 9th St. (16).

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Observations on the Use of Edathamil Disodium in Human Ears

JOSEPH SATALOFF, M.D., Philadelphia

A chelating agent is an organic compound capable of forming a stable complex with a multivalent positive ion such as calcium. In this process, coordinate bonding occurs in some of the atoms forming the ring, as a result of which the calcium becomes such an integral part of the complex that it loses its ionic properties and cannot be precipitated by commonly used precipitating agents.

This ability to bind calcium enables certain chelating agents to be used in the body to decalcify bone,[1] to remove calcium deposits in the cornea,[2] and to remove urinary calculi (experimentally).[3] Many new uses are now being reported. The agent is presently administered intravenously, and, if properly given, can produce good results without causing hypocalcemia or any other notable adverse effects in the body.

In exploring the use of a chelating agent in otosclerosis, at least four objectives become theoretically possible:

1. To make surgical mobilization of the stapes more certain by previous resolution of otosclerotic areas around the footplate with local application of a chelating agent
2. To prevent refixation of surgically mobilized footplates by intermittent or continuous application of the agent either locally or systemically
3. To cure otosclerosis by systemic administration of an agent that has specific affinity for otosclerotic areas
4. To prevent otosclerosis from developing in susceptible persons by systemic administration of a suitable chemical agent.

Enough work of a preliminary nature has been done to suggest the possibility of accomplishing at least the first objective.

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From the Department of Otolaryngology, Jefferson Medical College.

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During the past three years I have been investigating the effects of chelating agents in the ears of animals, cadavers, and patients with clinical otosclerosis. A previous report concerned itself with the effects of chelating agents upon stapes removed from human cadavers.[4] It was demonstrated that, by applying a chelating agent locally, the normal stapes footplate can be decalcified and softened without altering its shape or affecting the crura. The outer rim of the footplate was the first portion to be affected. Such findings gave hope of being able to soften otosclerotic areas around the footplate by applying a chelating agent locally to that surgical mobilization might become more certain in many cases and possible in some cases that are not now successful.

A number of chelating agents were evaluated, and edathamil disodium (disodium ethylenediaminetetraacetic acid) was selected as the most suitable for use in the middle ear. This agent was then studied at various concentrations and pH and with different vehicles to determine the solution most effective as a decalcifier and least irritating in the middle ear. The concentrations tested were 1%, 2%, 5% and 10%, and pH was varied from 5 to about 8. The vehicles used included normal saline, an oxycholesterol-petrolatum ointment base (Aquaphor), and polyvinylpyrrolidone (PVP).

The decalcifying effects were evaluated roughly by finding the time each solution took to soften a stapes footplate removed from a fresh cadaver. The effects upon 25 human middle ears were evaluated by applying the various solutions through large perforations in the tympanic membranes or by wrapping saturated wicks around exposed ossicles. At first only small amounts of the

agent were touched to the promontory wall, but later the entire middle ear was filled with certain solutions when it became evident that very few adverse effects were produced if the proper pH was maintained.

The most effective and least irritative solution so far utilized is 5% edathamil disodium at a pH of 7.3 or 7.4. This solution of edathamil disodium was then used in more than 30 ears of living patients with otosclerosis to investigate further any effects upon the middle and inner ear and particularly upon otosclerotic areas of bone.

Observations were made during stapes mobilization surgery, using a Zeiss operating microscope. The exposed middle ears of more than 12 patients were filled with edathamil disodium for periods from 10 minutes to 24 hours. (The latter was accomplished by intermittent injection through a plastic tube left under the skin flap.) Direct examination of the treated middle ears after 24 hours' exposure was done by lifting the flap.

In practically none of the patients was there any significant injection of the mucosa even after 24 hours' exposure. There was also no evidence of vertigo or nystagmus even when pledgets soaked in the solution were packed as deeply as possible into the round window niche. It is not yet established whether this apparently innocuous effect in these specific cases was because edathamil disodium is harmless to the labyrinth or because it was not transmitted through the mucosa of the middle ear or round window membrane. Efforts to establish the latter in animals and man are now also under way. In my experience, this particular solution of edathamil disodium has practically no notable adverse effect upon the mucosa of the middle ear except to cause a thickening after long exposure. Its effect upon the open labyrinth is still under investigation in animals. It can be stated, however, that solutions that are much lower than 7.3 in pH can result in a marked labyrinthitis. Since autoclaving may change the pH, it is essential that it be measured after sterilization. Until more information is available concerning the effects of chelating agents upon the labyrinths of animals, such agents should not be used indiscriminately in middle ears where there is a defect in the footplate or where perilymph is visible.

Edathamil disodium seemed to be more effective when the mucosa was removed from the exposed bone than when left intact. In several patients on whom it was not possible to mobilize the stapes because of excessive fixation, a small piece of absorbable gelatin sponge (Gelfoam) or absorbent cotton was placed directly over the footplates without removing the mucosa and then kept soaked with edathamil disodium for approximately 24 hours by means of a polyethylene tube extending into the external canal under the eardrum flap. When the gelatin sponge or cotton was removed and the ears examined, a slight thickening of the mucosa without significant injection was noted, but there was little noticeable effect upon the underlying bone as compared with the effects obtained when the mucosa was removed and the exposure repeated. (In performing such experiments, care was taken to remove all of the visible fibers of absorbent cotton.) While I believe it is preferable to have the solution in direct contact with the bony footplate and nearby otosclerotic area, it is quite possible that in some ears the mucosa around the footplate is so thin that it may readily permit the solution to act upon the underlying bone.

In order to compare the effect of edathamil disodium on otosclerotic bone with that on normal bone, five patients were selected who had not responded to surgical stapes mobilization because of overwhelming fixation of the footplates. In all these ears the heads of the stapes were disarticulated and removed along with the crura, permitting unobstructed views of the footplate regions. In two ears the mucosa was removed from the anterior half of the footplates and also from the nearby promontories and the otosclerotic areas anterior to the anterior crura. In two ears the mucosa was removed from the entire footplates as well as from the promontories and anterior otosclerotic areas.

In one ear the mucosa over the promontory was not removed. Wicks of cotton saturated with edathamil disodium were then placed upon the exposed bone and kept saturated for 24 hours through a polyethylene tube.

Examination of these ears after 24 hours' exposure clearly indicated that otosclerotic areas are much more affected by edathamil disodium than is normal bone. In all instances, the areas that appeared otosclerotic and were previously hard, resisting firm penetration with the pick or even chisel, now seemed much softer. The top layers could be peeled or chipped off like peeling a thick skin from an orange. The adjacent normal bone, on the other hand, was comparatively little affected. None of these patients experienced any vertigo or untoward reactions, except that two patients complained of a bitter taste in the throat when excessive amounts were introduced through the polyethylene tube.

In one of the patients the ear was reoperated on one month after treatment with edathamil disodium, and the area exposed was found to be still soft and easy to pick out. It was readily possible to penetrate into the oval window area and scrape out a reasonably adequate area into which a mucosal covering and an artificial stapes-like prosthesis could be inserted and attached to the incus. The promontory was not visibly affected. In another patient the ear was reoperated on six months later. While there seemed to be more firmness in the area than had been found immediately after exposure, it was still possible to create an oval window by gentle picking. It is probable, however, that the area was recalcifying.

While I have had a number of patients whose hearing has been improved by mobilizing the softened footplate after applying edathamil disodium locally, audiograms and case histories are intentionally omitted from this report. All my studies to date have been in the nature of pilot experiments, and there is no intention of advocating this solution as a tested therapeutic agent for otosclerosis at the present time.

Summary and Conclusions

After a series of pilot studies on animals, on cadavers and on living patients, over a three-year period, the chelating agent that seemed most effective and least irritating was a 5% solution of edathamil disodium at a pH of 7.3 or 7.4.

Results were better when the mucosa was removed than when it was left intact.

Otosclerotic areas seemed much more affected than normal bone.

Much additional work is needed before edathamil disodium, or any other chelating agent, can be advocated as a workable therapeutic agent in the treatment of otosclerosis.

The following during the past three years have significantly aided this investigation: Dr. John Zapp, Dr. Fred Harbert, Dr. H. Menduke, The Iso Sol Company, Mr. Philip Van Itallie, Dr. M. Seven, Dr. R. Gwinn, and Mr. Joseph Rosengarten.

1721 Pine St. (3).

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Laryngeal Vibrations: Measurements of the Glottic Wave

Part II—Physiologic Variations

ROLF TIMCKE, D.Sc.; HANS von LEDEN, M.D., and PAUL MOORE, Ph.D., Chicago

Part I of this report on Laryngeal Vibrations, which was published recently in this journal,[3] presented detailed measurements of the glottic wave for the normal vibratory cycle. For this study, a series of normal subjects were photographed with an ultra-high-speed Fastax camera during controlled episodes of phonation, and these films were subjected to frame-by-frame analysis. Selected pictures from typical scenes were enlarged for accurate measurements, and a number of interesting new observations regarding the normal vibratory cycle were recorded. The published data include quantitative information regarding the influence of intensity, pitch, and register on the vibratory pattern.

The results were transcribed graphically in norm measures as indicated on Figure 1. The terms "open quotient" and "speed quotient" * were employed to describe the relationship between the different phases of the vocal cord movements in each cycle. For a review of the pertinent literature, and for additional details regarding the technique, findings, and conclusions of this investigation, the interested reader is referred to Part I of this article.

Physiologic Variations

Part II of our report again presumes a normal organ of voice production and normal function. Eight representative graphs are presented to demonstrate some of the extensive and varied vibratory patterns which form the vocal response of the larynx to the exigencies of our daily life. As in Part I, the material has been divided into sections, and each group is analyzed separately. A comparison of each graph with

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Dr. Timcke now residing in Hamburg, Germany.

From the Laryngeal Research Laboratory of the William and Harriet Gould Foundation and Northwestern University Medical School.

* Open Quotient:

$$\text{O. Q.} = \frac{\text{Fraction of cycle during which glottis is open}}{\text{Duration of entire cycle}}$$

Speed Quotient:

$$\text{S. Q.} = \frac{\text{Time of abduction or lateral excursion}}{\text{Time of adduction or medial excursion}}$$

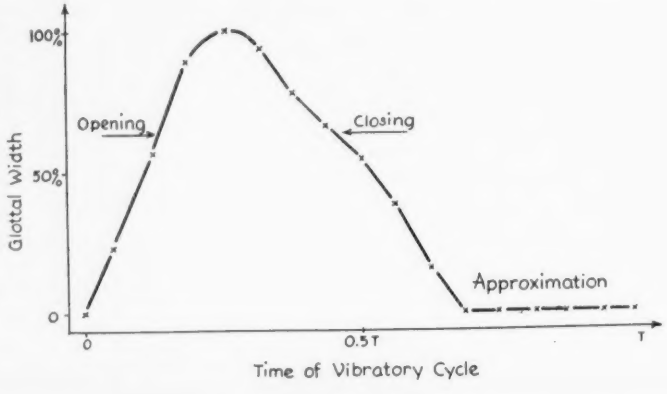

Fig. 1. — The vertical axis, or glottal width, represents the lateral excursion of one vocal cord as percentage of maximal opening. The horizontal axis indicates the time (T) in fractions of one vibratory cycle. Measurements from successive frames are indicated by X marks within the curve. The rising section of the curve depicts the opening phase; the declining section the closing phase, and the horizontal section the period of approximation.

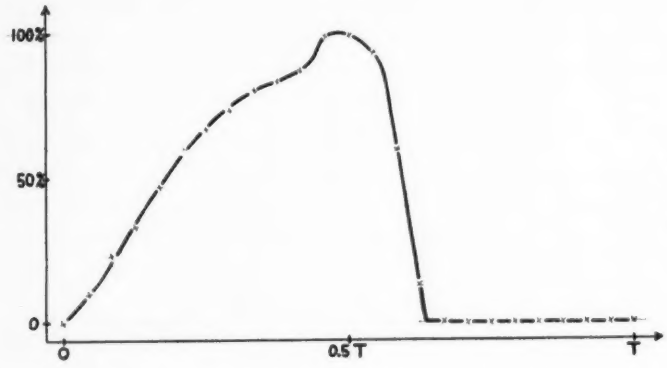

Fig. 2. — Subject T. Phonation: inspiratory. Register: chest. Pitch: low. Intensity: weak. O. Q.: 0.63. S. Q.: 3.2.

the normal vibratory cycle in the chest register and at medium pitch and intensity (Fig. 1) reveals the significant findings.

A. Inspiratory Phonation.—Figure 2 is typical of easy inspiratory phonation at low intensity. While this type of sound is not commonly heard per se, it is frequently produced during laughter, crying, or other emotional vocal manifestations. The arytenoid cartilages do not approximate fully in this type of phonation, but leave a small triangular opening posteriorly, similar to that observed during a stage whisper.

Speed Quotient: The most pronounced characteristic of this curve is the striking relationship between the opening and closing phases. It is apparent that the vocal cords separate (or abduct) much more slowly than they close (or adduct). Quantitatively speaking, this configuration results in an extremely high S. Q. of 3.2.

Opening Phase: This portion of the glottic wave presents a fairly well-defined convex hump. As indicated in Part I, this finding is commonly observed during the slower phase of the vibratory excursion.

Closing Phase: After a gradual acceleration during the early part of this phase, the speed remains almost constant until the cords have reached the midline.

Period of Approximation: In inspiratory phonation, the period of approximation is relatively long. In this curve the O. Q. measures only 0.63.

At first glance, these findings of a very high S. Q. and a relatively low O. Q. at weak intensity appear to violate the concepts which were so convincingly demonstrated in Part I of this article for the normal vibratory cycle. These data proved a direct relationship between intensity of phonation and speed quotient in normal voice production, and an inverse ratio between loudness of voice and opening quotient. In other words, weak intensity should be associated with a *low* S. Q. and a relatively *high* O. Q.

This apparent contradiction is readily explained by a brief reference to the aerodynamic principles which distinguish inspiratory phonation from ordinary sound production. In the former, two different factors supplement each other: the sucking effect of the Bernoulli phenomenon, and the closing effect produced by the anatomical configuration of the vocal folds. Because of the shape of these structures, static air pressure above the cords in inspiratory phonation tends to force the cords together. In contrast, the subglottic pressure of normal, expiratory phonation produces the opposite effect—opening of the glottis.

B. Low Harsh Voice.—Figures 3 and 4 are representative of the vibratory cycle during the production of low, harsh sounds. In these cases the harsh sound was produced deliberately and in a sustained manner, which is sometimes described as a "vocal fry." The harsh quality of the sound appears to be caused by a parallel tone of low frequency, which is audible in conjunction with the primary tone. Electroacoustic

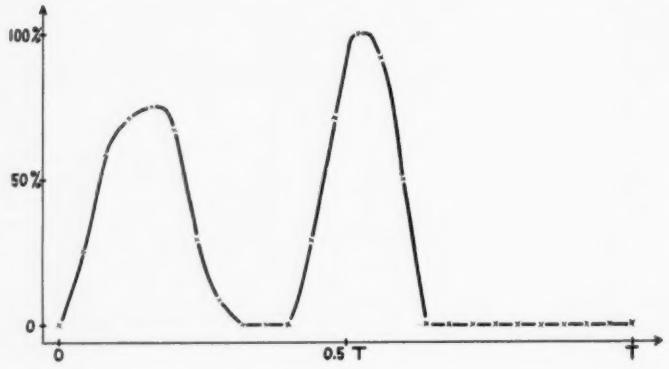

Fig. 3. — Subject T. Phonation: "vocal fry." Register: chest. Pitch: low. Intensity: weak. O. Q.: 0.8 and 0.4. S. Q.: 1.0 and 1.0.

measurements reveal the frequency of this low additional tone to be one-half the frequency of the fundamental pitch, which is associated with the specific sound under investigation.

On the basis of further studies,[1] it is our opinion that this type of phonation is often erroneously diagnosed by the clinician as ventricular dysphonia.

General Outline: The shape of these curves is striking and extraordinary. Each graph depicts two openings of the glottis during one vibratory cycle, and the configuration of these two movements differs significantly.

An extensive investigation of the pertinent film strips and the associated recordings confirms the physical accuracy of these graphs. While at first inspection each glottal opening appears like a complete vibratory cycle, closer study reveals that alternating vibrations are similar in appearance and that each set of two movements is followed by a prolonged period of approximation. In other words, the vibratory pattern changes periodically, the factor in this instance being 2.

Electroacoustic measurements of the sound recording accompanying Figure 3 indicate that the sound had a frequency of approximately 150 cps before the change to the "vocal fry" occurred. The length of the complete vibratory cycle on the graph, however, corresponds to a frequency of 75 cps, evidence that the strident quality of the sound is related to a "subharmonic" of the fundamental tone.

Amplitude: The maximal amplitude, or widest opening of the glottis, differs substantially between the two vibrations on each graph. In Figure 3 the amplitude of the first excursion is 25% smaller than that

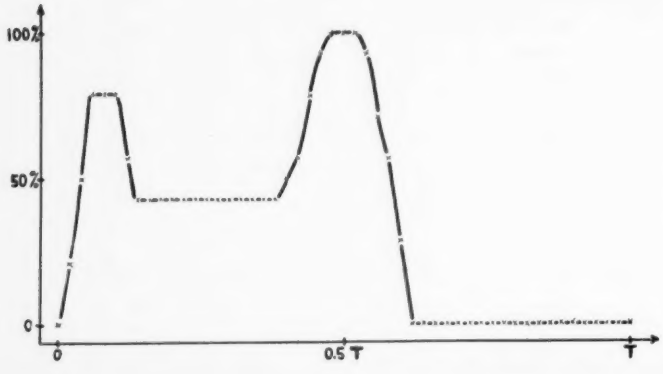

Fig. 4. — Subject B. Phonation: "vocal fry." Register: chest. Pitch: low. Intensity: medium.

of the second glottal opening. In Figure 4 the second vibratory movement again exceeds the first by more than 20%.

Speed Quotient: In spite of the different amplitudes, it is of interest to note that the speed quotients of the two vibrations in Figure 3 are equal: in each case the S. Q. measures 1.

In Figure 4 there is again a surprising similarity between the opening and closing phases of each vibratory movement, particularly if the asymmetry of this curve is taken into consideration.

Period of Approximation: Both graphs show a significantly different duration of the respective periods of approximation which follow the individual vibratory movements. In each instance, the first excursion is followed by a relatively short period of approximation, while the second period of approximation takes up almost two-fifths of the entire combined cycle. Thus, the measurements for the open quotient in Figure 2 are 0.80 and 0.40 respectively, which produce the clinical impression of a syncopated beat on the motion pictures.

Figure 4 presents a slightly different picture, because the vocal cords do not approximate fully at the completion of the first glottal opening. This asymmetry renders quantitative measurements of the open quotient unsatisfactory. However, clinically, this same arrhythmia produces an even greater feeling of syncopation during the projection of the film strips.[4]

Top of the Curves—Longitudinal Waves: While the two vibrations in Figure 3 show a soft rounded top, the two curves in Figure 4 present flat, even plateaus. As indicated in Part I of this article, these plateaus are the result of "longitudinal opening waves" which travel along the length of the vocal cords. It is our impression that the strong longitudinal opening waves in this case are at least partly responsible for the marked variance in pattern.

These examples prove conclusively that human vocal cords are capable of providing multiphasic patterns of vibration. They thereby confirm an earlier supposition of one of us (R. T.[2]), who had discovered a periodic change of the vibratory pattern on an artificial larynx (Helmholtz type) with certain differences in tension between the two artificial "vocal cords."

C. Staccato—Transitional.—A transitional, harsh sound produced involuntarily during the emission of several, sharp, staccato tones, is pictured graphically in Figure 5. The time of this phenomenon was too short to permit accurate acoustic measurements of its components, but the fundamental staccato note carried a frequency of just under 200 cps.

General Outline: It is readily apparent that Figure 5 portrays the same biphasic pattern of vibration as the previous two graphs. However, in this instance, the similarity between the two vibrations in each

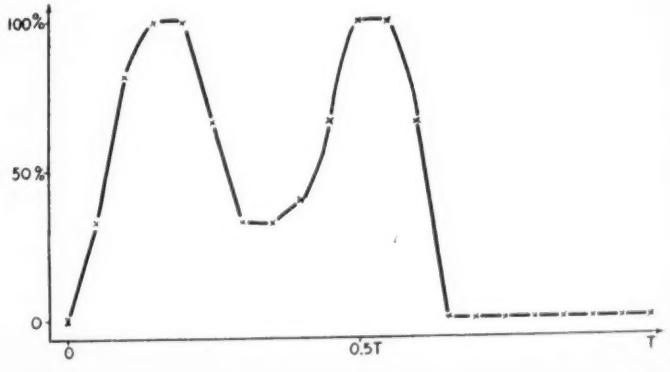

Fig. 5 — Subject S. Phonation: staccato. Register: chest. Pitch: low. Intensity: medium.

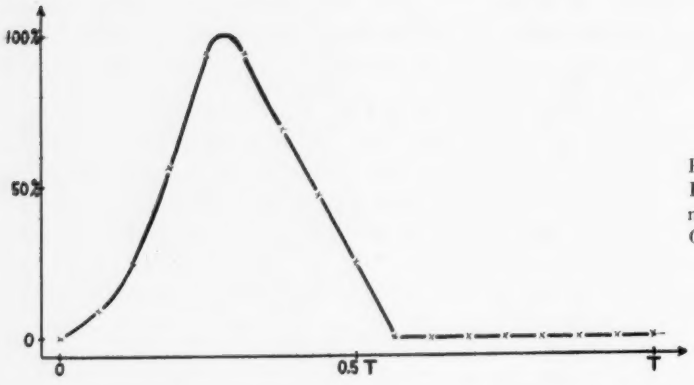

Fig. 6. — Subject T. Phonation: laughter. Register: chest. Pitch: medium. Intensity: weak. O. Q.: 0.55. S. Q.: 1.0.

cycle is much more striking, so much so that the two peaks are almost identical in configuration.

Amplitude: The two separate excursions show exactly the same amplitude.

Speed Quotient: The relative speeds of the two opening and closing phases almost duplicate each other. This similarity is all the more remarkable since the vocal cords do not return all the way to the midline at the completion of the first vibratory movement.

Period of Approximation: The asymmetry of the vibratory pattern does not permit quantitative measurements of the open quotient. However, the syncopated pattern of the vibratory cycle is plainly evident, with the second period of approximation consuming over one-third of the total cycle.

This graphic illustration indicates beyond doubt that periodic changes of the vibratory pattern occur involuntarily during the manifold laryngeal adjustments of our daily existence. Further studies are in progress to determine the physical and acoustic significance of this phenomenon.

D. Laughter.—Figure 6 depicts one vibratory cycle from a peal of light laughter. While laughter contains both expiratory and inspiratory elements, this scene was selected from the expiratory sequence.

Speed Quotient: In this instance, the vocal cords abduct at the same rate at which they return to the midline—a speed quotient of 1.

Period of Approximation: This phase of the vibratory cycle is quite long, with an open quotient of 0.55.

While the accompanying sound recording identifies this sequence definitely as an easy laughter, this graph pictures the real action in the larynx. As indicated by the fairly

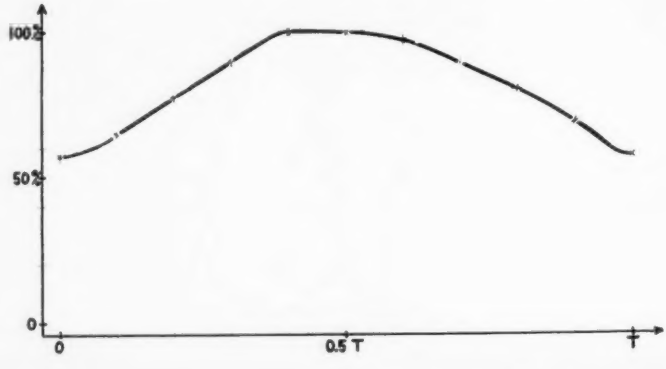

Fig. 7. — Subject M. Phonation: cough. Register: chest. O. Q.: (1.0). S. Q.: 0.67.

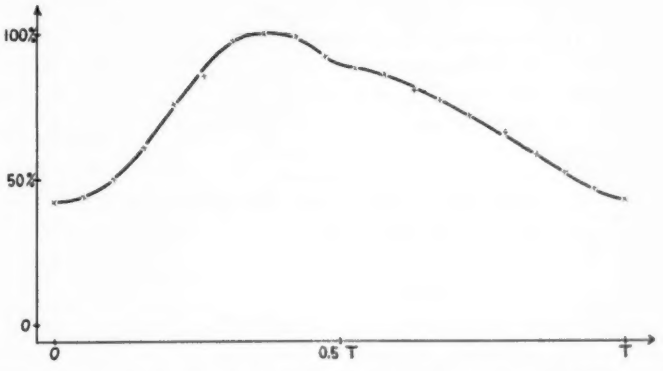

Fig. 8. — Subject F. Phonation: *coup de glotte*. Register: chest. Pitch: medium. Intensity: medium. O. Q.: (1.0). S. Q.: 0.59.

high speed quotient and the low open quotient, the vocal cords were moved by a force of at least medium intensity. These measurements emphasize the observation of vigorous changes in the larynx noted in ultra-slow-motion pictures during even the mildest laughter or clearing of the throat.

E. Abduction.—During sustained phonation it is, of course, impossible to produce vibrations with the cords in a position of abduction. To permit this type of investigation, a photographic trick was employed. Taking advantage of our ultra-high-speed camera and the resultant ultra-slow-motion pictures, we studied the vibratory pattern of the vocal cords toward the end of certain explosive sounds. At this point of the action, the vibrating vocal cords had separated in abduction, but had not yet come to rest. Examples of this type are the following: a cough (Fig. 7), a *coup de glotte* (Fig. 8), and a stressed termination (or *corpetto de voce*) (Fig. 9).

General Outline: The general outline of all three graphs is quite similar. In each case the vibratory excursion is confined to the lateral parts of the glottal opening, with gentle slopes representing the subsiding movements of the vocal cords.

Speed Quotient: The speed quotients are quite similar, varying from 0.67 in Figure 7 to 0.65 in Figure 9, and 0.59 in Figure 8.

Closing Phase: The closing phase of each graph again presents a poorly defined convex hump characteristic of the slower phase of the vibratory excursion.

Period of Approximation: Since the vocal cords do not approximate on any of these graphs, the open quotient has, of course, the fictitious value of 1. In Figure 9, there is a slight dip at the end of the vibratory cycle, which represents a weak flutter of the flaccid vocal cord.

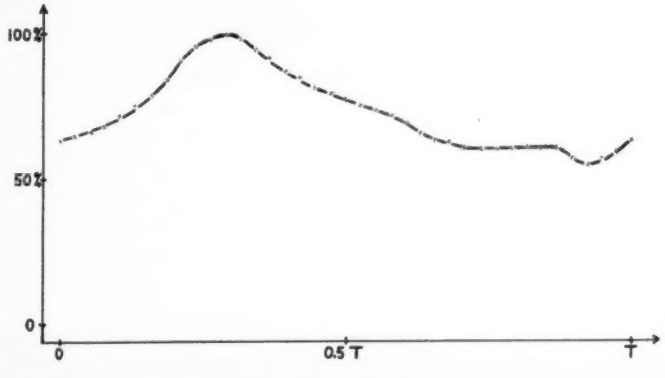

Fig. 9. — Subject S. Phonation: stressed termination. Register: chest. Pitch: medium. Intensity: medium. O. Q.: (1.0). S. Q.: 0.65.

Again, the speed quotient in this group of cases requires additional comment. While the values of the speed quotient in this group are quite low, they are not as low as one would anticipate from the great relaxation of the vocal cords. The explanation lies in the reduced influence of the subglottic air pressure, which differs markedly from that of sustained phonation.

Conclusions

1. *Structure of Vocal Cords.*—The anatomical configuration of the vocal cords plays a significant role in the production of the vibratory pattern, as demonstrated by the example of inspiratory phonation.

2. *Air Pressure.*—Changes in air pressure, both static and dynamic, exert considerable influence on the individual components of the vibratory cycle.

3. *Multiphasic Vibrations.* — Periodic changes of the vibratory pattern occur during voice production. Biphasic vibrations are typical of low, harsh sounds, which contain a strong subharmonic, in the described cases one-half the frequency of the fundamental wave. Transitional periodic changes also occur during complex laryngeal adjustments.

4. *Explosive Sounds.*—Laughter and other explosive phenomena, such as clearing of the throat, require a more forceful effort than their sound effect would indicate.

Results of Investigation

Previous observations regarding the effect of air pressure and structural changes upon the vibratory pattern were confirmed.

The wide range of laryngeal adjustments to the demands of daily use are stressed.

New observations are presented regarding the occurrence of multiphasic vibrations during normal voice production. A thorough search of the literature reveals no reports of similar observations.

30 N. Michigan Ave. (Dr. von Leden).

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Electrocutaneous Perception of Speech Sounds

MAX NELSON, Ph.D., East Lansing, Mich.

There is a possibility that within the cutaneous sensory receptors lies a means of aiding the deaf in the understanding and production of speech. The theory for this is that sounds have their own inherent characteristic patterns and will feel different or can be altered in such a way that they will feel different to the touch of the observer.

The purpose of this study was to determine the effectiveness of differentiating vowels cutaneously by electrodes and to compare a 1-electrode transducer with a 2-electrode transducer.

Background

Previous Studies.—In 1947, Anderson [1] conducted a series of exploratory tests in which several subjects participated. He reported that it was possible to feel differences between such words as Joe, toe, sew, how, now, cow, blue, and new. These tests were of a qualitative nature.

In another series of tests, Anderson utilized the vowel and consonant word lists of Fletcher and Steinberg.[2] The conclusion from this testing activity was that real discrimination resulted. In a parallel field of vibrotactility, R. H. Gault [3] and others conducted experimentation from 1924 through 1937.

Preliminary Experimentation.—The plan of experimentation was to record signals on a magnetic tape recorder and transmit these signals to subjects wearing a 1- or 2-electrode unit for tests of cutaneous differentiation. Vowel sounds were used as speech signals. The vowels were subjected to a process of compression in order to make them suitable for electrocutaneous reception.

The experimental design required that strict controls be exercised in the production and recording of the vowels, permitting the only varying factor to be the nature of the vowel.

Design of Equipment.—Four pieces of equipment were built specifically for this experimentation. These were as follows: the coaxial electrode, an electronic timer device, a 1-electrode amplifier, and a 2-electrode amplifier.

The coaxial electrode was constructed of brass and set in a rubberized plastic composition which had canvas ties at either end for fastening to a skin area. The inner conductor had a diameter of ¼ in.; the outermost dimension of the outer conductor was ¾ in.; the intervening space between the two conductors was 5/32 in.

The 1-electrode unit was a conventional amplifier and was constructed so that it responded to the range lying between 100 cps and 4,000 cps. A safety factor was incorporated to cut off transient peaks which might be painful.

The 2-electrode unit was constructed so that one electrode received frequencies between 100 cps and 800 cps while the other electrode responded to those frequencies between 800 cps and 4,000 cps. The 800 cps cross-over in frequency was based on

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This study was the doctoral dissertation for the Ph.D. degree granted by the University of Michigan in 1950.

vowel energy concentration research done with the sound spectrograph.[4]

The electronic timer device permitted the selection of two electrical signals in any combination for comparison tests. The length of the signal, the spacing between two signals, and the period between successive intervals could all be varied independently of each other.

The other equipment and accessories used were standard laboratory equipment.

Recording Process.—The process of recording a suitable speech signal on tape presented a major problem. In addition, an evaluation had to be made of the most effective manner of presenting the voice for electrocutaneous reception by the skin.

A series of exploratory tests was conducted to determine what changes should be made in the signal, what the duration of the signal should be, and what method of intensity control should be used.

Modification of Voice Signals.—Natural voice was used at first in the utterance of vowel sounds. This resulted in a cutaneous sensation which was generally unpleasant because of the presence of sharp points within the signal.

Next, the natural voice was subjected to compression and to an equalization process. Compression reduces the volume range by "pushing" together low- and high-level signals. Speech equalization boosts the high frequencies since normally the low frequencies are stronger. This method proved to be undesirable because of the unpleasant feeling that it caused. The unpleasantness of feeling was the reason for not trying speech equalization alone.

Finally, a third method was tried in which the speech equalizer was removed but compression was retained. This process proved to be the most effective from the standpoint of test results and subjective feelings.

Time Control.—Preliminary experimentation was conducted in order to determine the optimum length of the signals. As a result, an electronic device was built which automatically performed the following duties:

1. It removed the initial and final aspects of the vowel sound. This was done to eliminate clues which, although present in the onset and cessation of a sound, might not be inherent in the vowels themselves.

2. It controlled the duration of a signal, the time interval between two signals, and the time between pairs of signals. The timing of a single pair of signals was as follows: vowel 1—0.5 second, silence—0.3 second; vowel 2—0.5 second, silence—2 seconds.

Intensity Control.—It was necessary to determine how the skin responds to this kind of stimulation in order to devise an effective means of intensity control for skin stimulation. Two methods of intensity control were tried out. One method was that of equating the peak-to-peak amplitude of a sound wave by means of the oscilloscope. The other method, the root mean square, is a technique of equating sounds by determining the average power in them. This was accomplished by using a properly calibrated voltmeter. Experimentation indicated that the peak-to-peak amplitude method was the more effective means of intensity control. It more closely approximated the results of subjective judgments that had been made by the subjects.

Procedure

Subjects.—Ten normal persons participated, eight female and two male. They were all college students or college graduates.

Materials and Testing Procedures.—The vowels used were those in the following words: meet, mit, mate, met, mat, father (first vowel), all, obey (first vowel), cook, and cool. These vowels were divided into two comparable groups. The vowels within each group were combined into all possible pairs.

The procedure was for the experimenter to work with two groups of subjects consisting of five people in each group. Each subject experienced the 1-electrode unit and the 2-electrode unit an equal number of times of the total of 20 situations. Each daily meeting consisted of a practice session of 15 minutes which was followed by three tests of 2 minutes each. If, according to a prearranged order of randomization, the 1-electrode unit was to be used, it was placed on the dorsal area of the left forearm of the subject, midway between the

elbow and the wrist. If the 2-electrode unit was to be used, it was attached to the same area but separated a definite distance. Electrode paste was used to lower skin resistance. It consisted of sodium chloride, potassium bitartrate, powdered pumice and surgical lubricant. The tape recorder was adjusted to transmit the prepared signals through an amplifier to the electrode and to the subject. The subject was able to regulate the intensity level of the signal at any time.

During the learning session, the experimenter, who monitored the proceedings with an earphone, would state whether the two signals had been the same or different. The subjects felt the signals, made their own decision, then heard the experimenter's answer.

During the test, the subject would indicate on a specially prepared test blank if the two vowels felt the same or differently. There were no replayings of the test recording, and no comments were made. After the test was completed, the subjects gave the test paper to the experimenter, who graded them in private.

Results

The data can be considered most effectively by asking the following questions:

1. Do all vowel sounds lend themselves readily to electrode differentiation?

2. Is the 2-electrode system superior to the 1-electrode system?

3. Do all subjects perform equally well with the 2-electrode system?

4. Do all subjects perform better on the 2-electrode system than on the 1-electrode system?

Vowel Comparison.—Certain vowel sounds apparently do not lend themselves readily to electrode differentiation. In using as a criterion the group average score for the 1-electrode system of 59 points (out of a possible 90) and the 2-electrode score of 68 points (out of a possible 90), it was evident that the vowels in six tests failed to measure up to either standard. These were the vowel pairs in tests number 6, 9, 10, 14, 16, and 18 (all—at, at—ate, father—obey, obey—cool, ate—eat, and egg—obey). Test 15 (cool—in) was above the 1-electrode average with a score of 61 points but was 11 points below the 2-electrode average. Test 18 (egg—obey) responded poorly to the 1-electrode system with a score of 44 points. They improved by 23 points when used with the 2-electrode system. These sounds are separated by three vowels (at, father, all) on the acoustic continuum.* Vowels (cool—it) of Test 15 dropped from a score of 61 points on the 1-electrode system to 57 points on the 2-electrode system. This was difficult to understand since these sounds are at opposite ends of the acoustic continuum. The other five pairs of vowels (all—at, at—ate, father—obey, obey—cool, ate—eat) are located on the acoustic continuum within one sound of each other. This would seem to indicate that acoustic proximity would be a significant factor. However, this was not always the case. For example, the vowels (egg—father), which have one sound between them, responded to the 1-electrode system with a 68-point average while with the 2-electrode system it averaged 80 points. This condition prevailed also in the case of the vowels (book—all) in which the 1-electrode average was 66 points while the 2-electrode average was 82 points. It may be concluded that some vowels are more readily differentiable cutaneously than others. The reason for this is not apparent.

Electrode Comparison.—This was done by comparing the mean results of the same test for each of the two groups of subjects. In order for a real difference to exist, between the mean score of a vowel test on the 1-electrode system as compared with the mean score on a 2-electrode system, there must be a separation between scores of at least eight points.

Twelve of the twenty tests indicate a difference that is greater than the significant standard of eight points. In three other tests the average score was 78, 72, and 77 out of 90 for the 1-electrode system. These scores were above the group average of 59 points for the 1-electrode system by 19, 13, and 18 points. Thus, they should not be expected to improve as much as other subjects who had a lower performance level on the 1-electrode system. In regard to the 2-electrode system, the average score from each of these three tests was greater than

* Eat, it, ate, egg, at, father, all, obey, book, cool.

the group average of 68 points. These scores were 69, 76, and 70 points.

The remaining five tests resulted in low scores. However, the vowels of four of these tests were located on the acoustic continuum within one sound of each other.

Thus, the difficulty in differentiating these sounds was understandable. One puzzling problem was in Test 15 (cool—it). These sounds are at opposite ends of the vowel series. It would seem that with their acoustically dissimilar characteristics they would be quite differentiable. An additional point of interest was that all the average scores of the 2-electrode system were above the chance level of 45 points.

Individual Differences Between Subjects.

It appeared that differences did exist between subjects in the way they responded to electrocutaneous stimulation.

Difference in a Subject's Performance.—Seven of the ten subjects had a difference between their two scores that exceeded the standard of eight points. The differences between the scores of two of the subjects were not so great; however, the score in each instance for the 1-electrode system was so much higher than the group average that it partly compensated for this. The tenth subject did not show a real difference between her 1-electrode and 2-electrode scores; she did, however, have an average score for the 1-electrode system which was higher than the group average. No subject had an average score on the 2-electrode system which was as low as chance (45 points) or within the range of chance (35-54 points).

Summary

Ten normal hearing subjects underwent a series of 20 tests each. Each test was made up of two vowel sounds which had been compressed and were subjected to controls of duration and intensity. The vowels in each test were presented in a random order for a total of 90 combinations. The subjects made a judgment of same or different on each combination. Each subject used the 1-electrode system 10 times and the 2-electrode system 10 times. Both electrode systems were presented in random order. An aggregate of 200 tests, comprising 18,000 combinations of vowels, was administered throughout the experimentation.

Statistical tests, analysis of variance, were applied to the quantitative data. As a result of these analyses, the following conclusions would appear to be warranted:

1. Within the confines of the present study it can be stated that it is possible for most normal hearing subjects to differentiate controlled vowel sounds cutaneously.

2. The 2-electrode system is superior to the 1-electrode system.

3. Real differences exist between individuals in the way they perceive vowel sounds through the electrode systems.

Michigan State University.

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Acoustics of the Opened Periotic Vestibule

MERLE LAWRENCE, Ph.D.; ROBERT D. BURTON, M.D., and DAVID WOLSK, M.A., Ann Arbor, Mich.

On a purely logical basis it is obvious that when middle-ear adhesions prevent the ossicular chain from operating in a normal fashion the simplest procedure to follow is that of somehow breaking up the adhesions so that the mobility of the connection between tympanic membrane and oval window is restored. More than three-fourths of a century ago such techniques were tried with varying degrees of success. Speaking in terms of present-day audiometry, however, the techniques for measuring hearing were nowhere near as quantitative as those existing today and success was measured generally in terms of any noticeable improvement in hearing the spoken voice. Even with this generous (to the surgeon) method of determining "successful" surgery it was soon obvious that, to be specific, attempts to mobilize the stapes to restore hearing in cases of otosclerosis were not restoring hearing in more than about 25% of the patients. The "progress" of this form of surgery then went through a number of, now, very familiar steps. In 1897 Panse [1] described the use of chisels to loosen up the footplate of the stapes so that it might be removed. In a discussion of a later report by Panse,[2] Passow [3] suggested making a small hole in the wall of the labyrinth, and in 1898 Alderton [4] actually made holes in the footplate of the stapes. He found no improvement and advised against the operation.

This report is not a history of the development of operative procedures evolved for the relief of deafness caused by otosclerosis, mainly because such a review has already been done very adequately.[5] But the parallel between what went on more than 50 years ago and what is written today is alarmingly remarkable. In 1922 Fremel,[6] having assisted Bárány, reported results of opening a window in the posterior canal, and in 1923 Holmgren [7] reported on the use of a binocular microscope for making openings in the labyrinthine capsule. The final development was the highly successful procedure described by Lempert [8] of fenestrating the horizontal canal and reconstructing the middle-ear cavity so as to cover the new opening with a skin flap at the same time protecting the round window from direct sound.

Because of the success of the fenestration operation all others were abandoned until the stapes mobilization technique was reintroduced. As stated earlier, this procedure has the obvious advantage in certain ideal cases of restoring the ossicular chain to its normal condition, but unfortunately this is not always possible. The purpose of the investigation reported here is to provide an experimental basis for accepting or reject-

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Parts of this investigation were reported at the Symposium on Stapes Mobilization—Two Years Later, as part of the program of the 61st Annual Meeting of the American Laryngological, Rhinological and Otological Society, San Francisco, May 21-23, 1958.

From the Physiological Acoustics Laboratory, Department of Otolaryngology and Institute of Industrial Health, School of Medicine, University of Michigan. This investigation was supported in part by the Research and Development Division, Office of The Surgeon General, Department of the Army, under Contract No. DA-49-007-MD-634 and by funds for research in Human Resources, University of Michigan.

ing some of the history-repeating procedures of opening the perilymphatic space in the region of the stapes footplate. In the face of the successful and highly developed Lempert operation it would seem wise to put some faith in the judgment of the surgeons of 25 to 50 years ago unless experimental evidence proves otherwise.

This series of experiments was designed to explore these possibilities, using a technique that continuously monitors the cochlear potentials while various middle-ear manipulations are carried out.

Method

A speaker unit mounted outside a shielded, soundproofed room was driven in the usual way by an oscillator-attenuator-power amplifier system. A hard-rubber tube conducted the sound from the speaker unit through the shield and walls of the room to the animal-operating area. This tube was terminated by a small hard-rubber cannula which contained concentrically the probe tube of a calibrated WE 640AA condenser-microphone-probe-tube system which furnished the data for determining sound pressures. The cannula and its concentric probe tube were small enough to be tied into the external meatus of the animal so as to form a completely closed system with the speaker unit at the other end.

The cochlear responses of the animal were picked up by a platinum-foil electrode placed so as just to touch the center of the round-window membrane with an indifferent electrode in the temporalis muscle. These potentials were amplified exactly 1,000 times by a battery-operated amplifier and then conducted to a General Radio type 736-A Wave Analyzer outside the room. The wave analyzer acts as a selective voltmeter tunable to any frequency from 0 to 16,000 cps with a bandwidth of six cycles. It achieves this selectivity by combining the output of a variable frequency oscillator with the incoming signal so that the sum of the two is always at 50 kc. This signal is passed through crystal filters, rectified, and metered. By picking up the 50 kc. signal immediately after the filters it is possible to utilize practically the entire 60 db. range of the early analyzer stages. Amplifying this signal and sending it through a Sound Apparatus Company Model SL-4 recorder, the response from the animal can be monitored continuously with a usable range of 60 db. In addition to response-voltage measurements made at various frequencies in the usual way on the wave analyzer, the continuous-running responses for 1,000 and 2,000 cps were made on the recorder while various alterations of the middle-ear structures were surgically introduced.

Healthy, normal adult cats were used for these experiments. These were deeply anesthetized with a solution of diallylbarbituric acid with urethan administered intraperitoneally. The bulla was exposed and opened by a lateral approach, and the pinna was removed so as to leave about 10 mm. of external canal into which the sound tube with its concentric calibrating probe tube was tightly tied. Before any surgical alterations of the middle ear were undertaken a 3μv. sensitivity curve for frequencies between 100 and 10,000 cps was taken to serve as each cat's normal threshold for later comparisons.

Results

The results of disrupting the ossicular chain have been described many times both clinically and experimentally,[9] but, as a preliminary step in the procedure to follow, the incus in the first animal was removed. This was done by carefully dissecting around the tympanic annulus posteriorly until the stylomastoid foramen was opened and the facial nerve exposed. In the cat this foramen lies almost directly posterior to the annulus along a horizontal line drawn through the middle of the tympanic membrane. The bone of the region was nibbled away with a rongeur until the stapedius muscle tendon was exposed. This gives a good view of the entire middle ear, the stapes, and its footplate. In all operations a Zeiss operating microscope was used.

With a small knife the incudostapedial joint was severed. The fossa for the short process of the incus had already been exposed during the bone dissection, and now the tendons holding the short process were cut and the incus removed. The stapes was intact and undisturbed; the tympanic membrane was normal with the malleus attached, and the middle ear was clear of fluid and blood.

Figure 1 shows the audiogram of this cat as measured by the cochlear potentials. This is exactly the extent of loss that would be predicted from the many previous reports that have been made in cases where the ossicular chain has been disrupted. The average loss for all frequencies in this in-

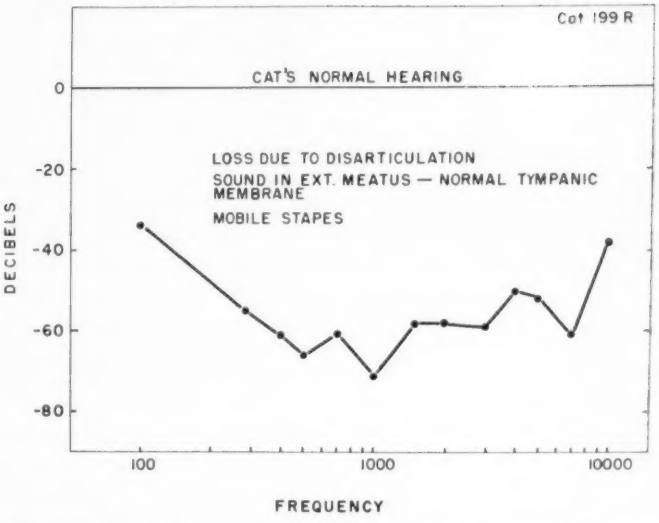

Fig. 1.—The loss in sensitivity produced in a cat's hearing by removal of the incus.

stance is 56 db. The explanation that has been given and confirmed for this much loss in these circumstances is that the drum membrane now serves no useful purpose, but acts as a barrier to the sound entering the external canal. In addition to this the sound has access to both the oval and round windows, which produces some canceling of inner-ear mobility.

The stapes was not fixed in this preparation; however, it is possible to determine the acoustic properties of the ear when the incus and stapes are removed completely, but the tympanic membrane remains intact.

Figure 2 is a running-time record for 1,000 cps taken on the animal of Figure 1 after the incus had been removed. It can be seen that removal of the stapes has a detri-

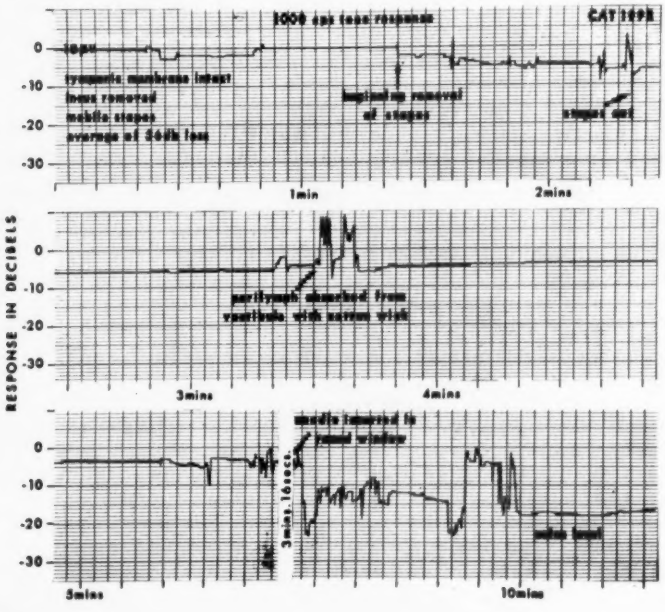

Fig. 2.—A running-time record of the same animal as Figure 1, showing the effect of stapes removal in an ear with disarticulated ossicular chain.

mental effect of only about 4 db. even with the perilymph removed from the vestibule. This was recorded merely over a six-minute period after removal of the stapes and does not show what might have happened to hearing over a longer period of time. A needle was then inserted into the round window to destroy the endolymphatic system in order to check that the recording was not in some way an artifact. Obviously it was not. This destruction reduced the potentials to the noise level of about 1 μv. The explanation of these results probably lies in the fact that with no middle ear the sound hits both windows a little out of phase because of their positions, and it makes no difference to the endolymphatic system on the oval window side whether the sound comes through a mobile stapes and the perilymph to the endolymph contents, or hits the membranous labyrinth directly. (Possibly bone conduction is also involved at the sound levels required under these conditions.)

In previous experiments it has been demonstrated that, in the absence of a middle ear, the possession of two open windows gives better hearing than that of one opened and the other blocked. It stands to reason then that, if the round window is normal and the stapes fixed, any sort of opening on the scala vestibuli side of the cochlear duct would improve hearing somewhat. This opening can be achieved by a fenestra in one of the canals, a fenestra in the footplate of the stapes, or even by removing the stapes. Obviously, however, without an ossicular chain or similar advantages achieved by plastic reconstruction such a situation does not produce normal hearing, merely hearing that is better than when only one window is mobile. Under certain circumstances, such as a better than normal inner ear, two unimpeded windows may produce near normal hearing, but the expected loss on the average would lie in the vicinity of 30 db. or greater. The question remains as to whether a hole or crack in the footplate of the stapes alters the characteristics of an already mobile stapes.

Figure 3 shows, in a second animal prepared in the same way as the first, that a hole in the footplate of a mobile stapes makes very little difference over a frequency range of 300 to 10,000 cps; in fact it makes the situation a little worse. The running-time record for this animal, Figure 4, shows the fenestra made at the end of 3 minutes 10 seconds. There was some bleeding and loss of perilymph when this was done, and removing this showed an audiometric loss of about 4 db. Removing the stapes completely and absorbing the released blood and perilymph made no immediate difference, but there was a gradual deterioration of the response over the next five minutes. Opening the vestibule either by perforating the footplate or removing the stapes has no advantages over a mobile stapes; in fact, it has only dangerous disadvantages because of the consequent loss of perilymph.

In a third animal simulation of an actual mobilization and subsequent vestibule opening were undertaken. This was done in the absence of an effective ossicular chain and followed by removal of the tympanic membrane.

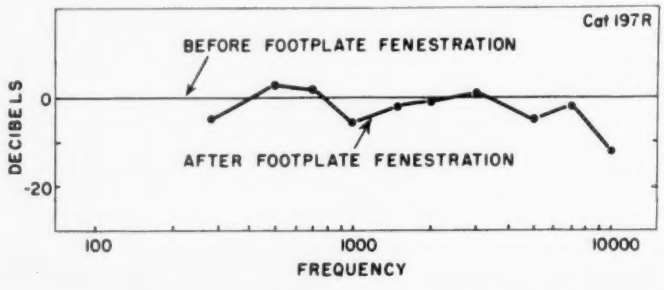

Fig. 3.—The effect of footplate perforation in a mobile stapes and with disarticulated ossicular chain.

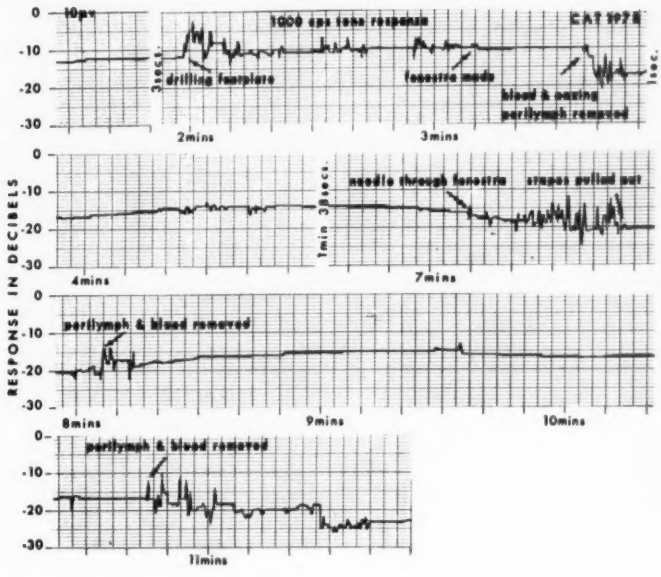

Fig. 4.—A running-time record of the same animal as Figure 3, showing the effect of drilling the footplate, the effect of removal of the stapes, and subsequent degeneration of response.

The stapes of this animal was first fixed in the presence of a normal ossicular chain by the pressure of a needle deeply and firmly embedded in the Eustachian tube. The crura of the stapes clear the promontory by only a fraction of a millimeter, and it is possible to wedge the stapes between an anchored needle and the promontory without disrupting the incudostapedial joint. The dashed line of Figure 5 shows the audiogram under these conditions. The average loss between the frequencies of 200 and 1,500 cps is 45 db. and that for the full range is 40 db.

After fixation of the stapes a running-time record was started with the response set at about 3μv. so as to keep the sound level as low as possible. The tone recorded was 2,000 cps, a frequency which, apparently, this kind of fixation did not hurt as much as the lower ones. This record is shown in Figure 6.

The first procedure was that of mobilizing the stapes. This was accomplished by frac-

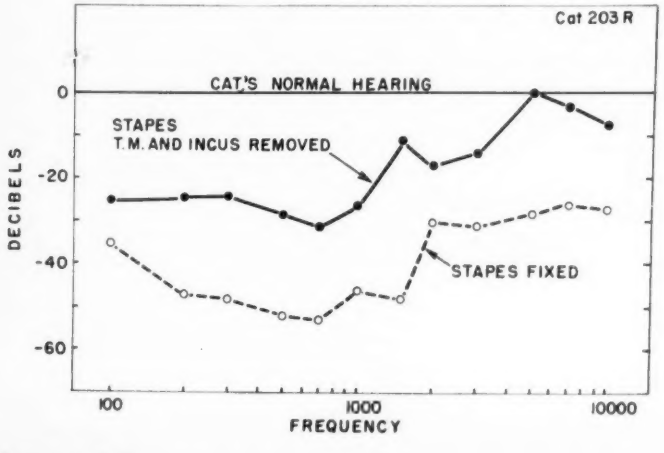

Fig. 5.—The effect of stapes fixation and the improvement subsequent to extirpation along with the tympanic membrane and incus. In view of the hazards and probable degeneration of the membranous labyrinth, the fenestration operation is recommended in place of any procedure that opens the vestibule.

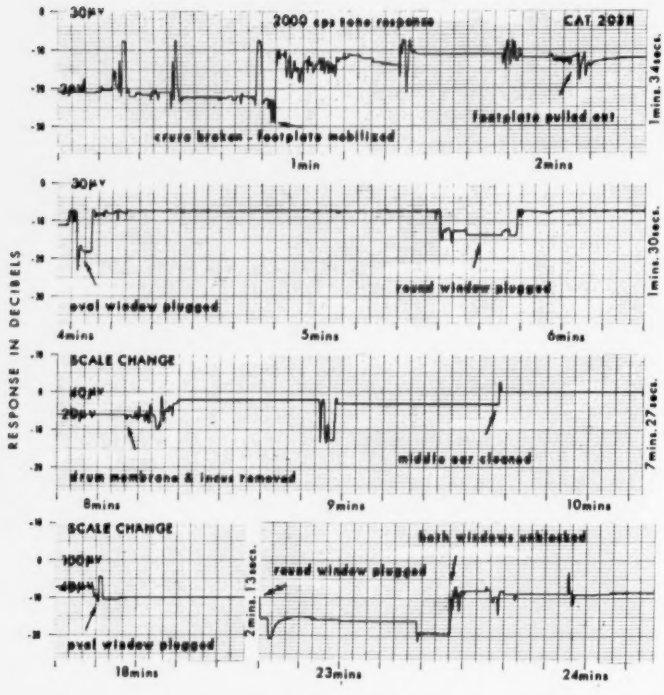

Fig. 6.—A running-time record of the events leading to the solid-line sensitivity curve of Figure 5. When either window is blocked under any conditions, the response drops.

turing both crura below the needle anchoring them. The record shows an immediate improvement in response of 12 db. Mobilizing the footplate in this manner has also the effect of disrupting the ossicular chain, but even in view of this there was an improvement. The footplate was then completely removed from the oval window, which, after the blood and escaping perilymph were cleaned up, produced a further improvement of about 3 db.

In order to check on the effectiveness of the oval window in favoring sound transmission under these circumstances the oval window was blocked as thoroughly as possible by packing it with bone wax. The result is shown on the record at the end of four minutes of running time. The response was reduced to within 3 db. of the level before mobilization. Opening the window brought the response up to 13μv., an improvement of 13 db. from the original condition of fixed stapes. At five and one-half minutes the round window was plugged with bone wax, producing a loss of 6.5 db. Removing the plug returned the response to normal, indicating that the freedom of both windows is essential for maximum hearing when the incus is removed. Further cleaning of the middle-ear cavity by removing blood and escaped perilymph improved the response to 20μv., more than 16 db. above the original. This necessitated a scale change on the running record and is so indicated. A little after eight minutes had passed, the tympanic membrane and incus were removed, thus taking out the useless barrier to sound which had been absorbing some of the sound and serving no useful acoustic purpose. Cleaning the middle ear of newly accumulated blood helped the response 3 db. more—now at 40μv., an improvement of 22 db. above that obtained in the original condition.

Both windows were now plugged. First, plugging up the oval window produced a 3 db. loss, and further plugging of the round window increased the loss to a total of 12

db. This is still more response than there was originally, and this probably reflects the ineffectiveness of the two window blocks. Since the sound level must, under these conditions, be raised to quite a high level, there may be some bone conduction response also present.

Unblocking of both windows returned the response almost to the 40μv. level again, and at this point another sensitivity determination was made. There is no indication that this situation would have lasted.

Referring again to Figure 5, the solid line shows the audiogram for 12 frequencies from 100 to 10,000 cps after mobilization of the stapes, in fact, after its complete removal, and after the tympanic membrane had been removed and the middle ear completely exposed. There is marked improvement even with the stapes removed and the perilymphatic spaces exposed, and the results shown here are better than would be expected on the basis of previous experimentation. It should be kept in mind, however, that these experiments are of short duration and give no indication of the long-term hazards of exposing the inner ear in this manner. Unless a skin flap were provided to cover the exposed labyrinth and to protect the round window from the sound source, this procedure of exposing the vestibule has only dangerous disadvantages.

Conclusions

It is an obvious fact that no surgical alterations of the ear can do a better job of sound conduction than a normal mechanism. The best that can be done is to restore the normal situation as nearly as possible by, in the case of an ankylosed stapes, restoring the normal mobility of the chain or at least establishing a mobile plunger of some sort still attached to an ossicular chain and occupying as much of the oval window as possible.

There is no experimental evidence, whatsoever, when conditions are carefully controlled, the physical arrangement of the middle ear accurately noted, and audiograms made with precisely calibrated equipment, that normal hearing persists when the ossicular chain is disrupted.[10] This is with a mobile stapes; and, if it is fixed, the situation is even worse.

In a middle ear that has no connected chain, i. e., has lost the incus, two open windows produce better hearing than when one is opened and the other closed, as would be the case with normal round window and fixed stapes, or with mobile stapes and occluded round window.

Perforation or removal of the footplate to free the oval window has advantages only when there is no continuity to the ossicular chain, but these procedures open the inner ear to infection and allow perilymph to escape. During the last two decades of the 1800's when the first cycle of stapes mobilization was at its peak, removal of the stapes was tried and abandoned for these very reasons.[11,12] If a mobile plunger in the oval window connected to an ossicular chain and intact tympanic membrane cannot be established, it is far better to do a fenestration, where good results are predictable and continuous loss of perilymph with possible degeneration of the membranous labyrinth is avoided by the presence of the trabecular system of the horizontal canal and the closing influence of a flap.

4506 Kresge Medical Research Building.

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Case Reports

Extramedullary Plasmacytoma of the Nasal Cavity and Lateral Wall of the Nose

R. J. McQUISTON, M.D., and D. E. JONES, M.D., Indianapolis

Extramedullary plasmacytoma is a very uncommon neoplasm in the nose and nasal accessory sinuses. This is a report of an unusual case, which we have followed for a period of 12 years, and in which we have operated twice, with a different pathological diagnosis each time.

This patient, a 63-year-old white man, was first seen about 12 years ago. He gave a history of being unable to breathe through the left side of his nose. Several years prior to this, he gave a history of having nasal polyps removed from this side. Examination, at this time, showed the left side of the nose to be obstructed with a smooth fibrous tumor, originating from the lateral wall of the nose and adhering to the nasal septum.

He was admitted to the hospital, and, under local anesthetic, the tumor was dissected free from the septum and the lateral wall of his nose and removed. The frozen section, at the time of surgery, was diagnosed as a adenocarcinoma. The permanent section, reported later, did not support this diagnosis; however, a positive diagnosis could not be made until a slide of the tissue was sent to several other pathological laboratories, and, after considerable study, the diagnosis of sarcoidosis of the wall of the nasal cavity was made.

The patient was examined at least once or twice a year for the following 12 years, without a recurrence of the obstruction or tumor until the last two years, when there was a gradual swelling, and deformity began to occur over the left lateral side of the nose externally. The tumor became a hard, firm mass, but was not painful. The patient ignored this slowly forming tumor, for some time, until it eventually became so large it obstructed the tear duct on the left side. His vision was partially obstructed owing to the tumor. He was admitted to the hospital for the second operation, and a complete physical examination was essentially negative except for the tumor on the left side of the nose.

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X-Ray Findings.—X-ray films of the chest showed that the diaphragm was smooth on each side, and the costophrenic angles were clear; except for a very small fibrocalcified lesion in the right upper lung field, the lungs were free of active inflammation or metastatic abnormalities. The ribs and sternum presented no evidence of metastatic disease or osteolytic defects. The thoracic spine in the lateral projection revealed no pathology.

AP, Towne, and Left Lateral Views of the Skull.—These showed the cranial vault was normal in size and dimensions. Prominent diploic markings were present bilaterally, but no definite osteolytic lesions of the vault or base of the skull could be seen. The sinuses were reported as essentially negative.

Operative Procedure.—With the patient under a general anesthetic of thiopental (Pentothal) sodium, a local infiltration of procaine, the patient was anesthetized. An incision was made along the base of the nose from the internal margin of the inner canthus of the orbit, down to the alar cartilage; the skin and superficial fascia were reflected, and a soft tissue tumor mass was found, which was very vascular in nature.

The tumor mass was resected loose, and it involved the lateral wall of the nose, which had been eroded by the tumor mass, and extended into the nasal cavity, involving the anterior ethmoid cells. This tumor mass was resected free, and the anterior ethmoid cells removed. The lacrimal sac was also involved by the pressure of the tumor mass, and the lacrimal bone under the lacrimal sac was removed and the opening made into the lacrimal sac to allow it to drain into the nose. After the tumor mass was exenterated, the nose was packed with one postnasal pack and three strips of 1 in. iodoform packing. The external wound was closed with plain No. 00 suture and black silk. He was given one blood transfusion, because of excessive bleeding during the surgery. The patient's condition upon completion of surgery was satisfactory.

Postoperative Diagnosis.—Soft tissue tumor mass involving the lateral wall of the nose, the

soft tissue of the skin and involving the ethmoid area.

Surgical Pathology.—Gross Examination: The tissue, which was studied by frozen section, was poorly circumscribed, and not encapsulated. The mass was grayish-white and rather fibrous in consistency.

Microscopic Examination.—Frozen Section Impression: Neoplasm, type to be determined. Histologic study shows the presence of a densely cellular neoplasm, which is extensively infiltrating the soft tissues of the nose. In the better preserved area, the cells are seen to be plasma cells, which are arranged in cohesive groupings. While these cells are identifiable as plasma cells, they differ from inflammatory plasma cells in having atypical nuclei, which vary in size and shape and in which the cartwheel nuclei are no longer preserved.

The cytoplasm is more abundant, and there are scattered mitotic figures. The tumor is moderately vascularized. It is poorly circumscribed, and sheets and strands and cords are extensively infiltrating outward into the soft tissue of the nares. The surrounding tissue shows fibrosis. Many pools of pink-staining hyaline material are noted within the tumor. This material is reminiscent of the type of protein seen in the renal tubules in cases of multiple myeloma. This type of material has stimulated a considerable foreign-body type reaction. In comparing this with previous surgery, received in 1946, the process is seen to be essentially similar, except that the plasma cell nature of the tumor is not as apparent in the biopsy 12 years ago.

The neoplasm is interpreted as an extramedullary plasmacytoma, a very uncommon neoplasm. The definite relationship between this neoplasm and multiple myeloma has never been definitely ascertained. Multiple myeloma should carefully be ruled out before the diagnosis of extramedullary plasmacytoma is established.

Permanent Section Impression: Extramedullary plasmacytoma, soft tissues, lateral wall of nose with associated acidophilic hyalinized material and secondary foreign-body reaction. This diagnosis was reached after considerable consultation with the pathology department, the tissue having been studied by several in the area. The diagnosis was plasmacytoma. This type of tumor is probably a benign tumor; however, it is closely associated with multiple myeloma, and in common with it may involve other areas of the body, such as the skull, the sternum, and other skeleton bones. It is not known just what relationship it has with multiple myeloma, but, according to the pathologist, the two are usually somewhat associated.

The patient made an uneventful recovery, with no deformity following the surgery. It was recommended that in the future this patient have periodic thorough physical examinations with particular attention to the blood studies and x-ray studies of his skull, chest, sternum, and other skeletal bones for evidence of an associated developing multiple myeloma.

608 Guaranty Building (4).

Chondroma of the Larynx

Report of a Case with Recurrence After Twenty-Eight Years

MAURICE SCHIFF, M.D., Oakland, Calif., and M. S. BENDER, M.D., St. Albans, N. Y.

Chondromas are among the rare connective-tissue tumors of the larynx.[1] Gatewood,[2] in 1942, reported two cases of this laryngeal tumor and brought the literature up to date with a total of 83. Since then several other cases have been published. Ryan and Zizmor[3] reported another in 1949, which demonstrated an additional roentgenographic technique for detecting these tumors. Link,[4] in 1949, augmented the total of reported cases of this tumor to 88.

The purpose of this paper is to add to the literature an unusual case in which a recurrence appeared 28 years after the original removal.

Etiology

Nothing is known as to the cause of chondromas of the larynx. However, it has been noted statistically that 80% are found in men, and the fifth decade is the most frequent period of occurrence.[5]

Classification

For practical purposes Tobeck's classification[6] is probably the most useful. He suggested the following: (1) chondromas arising from the inner surface of the larynx, and (2) those arising from the outer surface of the larynx.

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Consultant in Otolaryngology, U. S. Naval Hospital, St. Albans, N. Y. (Dr. Bender).

Captain, MC, U. S. N.; Chief of the Eye, Ear, Nose and Throat Service, U. S. Naval Hospital, Oakland, Calif. (Dr. Schiff).

The opinions or assertions contained herein are the private ones of the writers, and are not to be construed as official or necessarily reflecting the views of the Medical Department of the Navy or the Naval Service at large.

Symptoms

The symptoms depend upon the position and size of the tumor. If there is an encroachment upon the airway, the complaints will be referable to breathing. Hoarseness, cough, and a loss of voice may be present. If the tumor mass extends posteriorly towards the cervical esophagus, symptoms referable to swallowing develop. The last mentioned is a less common finding. Since the tumor growth is at a very slow rate, the symptomatology is insidious and frequently not observed until intercurrent infection or sudden demand from exertion precipitates the attack which brings the patient to the laryngologist.

Diagnosis

Because of the nature of this tumor and the late occurrence of complaints, a diagnosis is not usually made until the symptoms become urgent or an external mass appears on the larynx. Laryngoscopy, roentgenography, and physical examination are of considerable aid in establishing the diagnosis. To obtain a biopsy of this lesion endolaryngeally is difficult, as has been noted by others,[3,4] but it should be attempted whenever possible.

Report of Case

A 68-year-old white housewife was admitted with a chief complaint of hoarseness and occasional difficulty in breathing. She also had noted a small mass growing on the right side of her neck.

About 28 years previously the patient had a walnut-sized tumor removed from the throat. She did not know the type of tumor or its location, and the operative report of the original surgery was unobtainable. However, since then she had been hoarse, a condition which had become slightly

worse in the last two years. About two years prior to admission the patient noticed a nodule about the size of a small walnut in the right side of the neck at the site of the previous tumor, and during this time there was little change in the size of the mass. She had been experiencing shortness of breath for this length of time but attributed it to the high blood pressure for which she was under treatment.

The patient gave a history of having been hit with a baseball in the throat at the age of 21 years. This was 19 years prior to the first operation.

Physical examination showed a well-developed, slightly obese woman in no acute distress. There was a 2×2 cm., hard, smooth nodule which appeared to be fixed to the right side of the larynx and moved on swallowing; also a dense, old scar in the midline extending from the cricoid to the suprasternal notch. There were several small, soft, palpable lymph nodes in the right cervical chain. Indirect laryngoscopy revealed moderate edema of the entire laryngeal structures with marked distortion of the intrinsic larynx. The larynx appeared to be rotated, and beneath the distorted epiglottis there seemed to be an inward bulging of the right ventricular band. No true right vocal cord could be seen, nor was there any movement in the area of the right arytenoid. All phonation appeared to be made by the compensatory effort of the left vocal cord. Nowhere could any ulcerative discontinuity in the mucosa be seen.

Direct laryngoscopy confirmed the above findings, and, since no break in the mucosa was visible in the larynx or subglottic trachea, it was deemed best not to take a biopsy of the mass endoscopically.

The lungs were essentially normal. The heart was enlarged to the left; the pulse rate was 92 and the rhythm markedly irregular, owing to auricular fibrillation and flutter. No murmurs or thrills were present. The blood pressure was 160/80. The impression was arteriosclerotic, hypertensive, cardiovascular disease without decompensation. The remainder of the examination was essentially normal.

Roentgenographic examination showed a large amorphous calcific density in the region of the right thyroid cartilage (Figure). All anatomic relationships were lost as there was no semblance of cricoid or thyroid cartilage remaining in this area.

On June 29, 1950, under local anesthesia, a laryngofissure was performed and a firm, well-circumscribed mass was found to involve the major part of the right thyroid and the entire right cricoid cartilages. The mucosa of the larynx was separated from the mass, which was removed completely. In attempting to perform a tracheotomy prior to closing, it was discovered that the upper

Lateral view of the larynx showing the large amorphous calcific density involving the cricoid and thyroid cartilage.

three tracheal rings were deficient on the right. This was due either to erosion of the present tumor or this portion had been removed or involved in the tumor which was removed 28 years before. The failure of the right half of the larynx and trachea to collapse after the loss of so much supporting structure probably was the result of the large amount of fibrous tissue which had grown in this area during the interval between the two operations. A tracheotomy tube was inserted and the larynx closed in layers.

The specimen was submitted to the laboratory, and the microscopic examination revealed somewhat cellular cartilage which was uniform in appearance and contained no bizarre nuclei. Pathologic diagnosis: Chondroma.

Clinical Course.—The patient had a slightly febrile postoperative course for the first three days with much productive mucoid sputum. Following this period, recovery was prompt and she was decannulated 20 days after the operation and discharged home.

In monthly follow-up visits the patient was observed to have an adequate airway. She also thought she could speak with a louder hoarse whisper than was possible preoperatively. However, clinically there appeared to be very little change.

Comment

The case presented represents several unusual features which are interesting to observe. This patient undoubtedly had a local recurrence of a tumor 28 years after removal. After the first operation she developed hoarseness and had spoken with a harsh whisper ever since that time. The presence of such long-standing hoarseness probably dulls the natural sense of suspicion associated with this symptom. In addition, the slow, progressive development of arteriosclerotic, hypertensive cardiovascular disease gave adequate explanation for the dyspnea on exertion. Thus again, another cardinal symptom, dyspnea, was attributed to a systemic disease process and the usual alertness for a laryngeal cause was blunted. It was not until the mass on the side of the neck was observed that the presence of a laryngeal tumor was entertained.

The effect of local trauma, such as a baseball to the side of the neck, leaves room for much speculation. The interval of 15 years between the injury and the appearance of the nodule on the right side of the larynx for the first time might seem to be too long to show any causal relationship. However, since a chondroma is a very slow-growing tumor, taking more than 28 years in this case to show first signs of recurrence, the time factor might warrant reevaluation.

Summary and Conclusion

This is a somewhat unusual case of recurrence of a chondroma of the larynx with a history of local trauma 15 years prior to the first appearance of the tumor and an interval of 28 years until the recurrence.

Prompt surgical excision is the treatment of choice, as early removal may preserve at least part of the larynx.

U. S. Naval Hospital, Oakland 14, Calif.

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Malignant Granuloma of the Nose

PHILIP H. VON FRAENKEL, M.D., and WILLIAM J. HITSCHLER, M.D., Philadelphia

Midline lethal granuloma remains as much a mystery as when it was first described before the turn of the century.[1] A rare condition which usually starts in the nose, it spreads centrifugally, involving both superficial and deep nasal tissues. There is first local thickening and gradual interference with tissue nourishment and drainage. Ulceration may occur early or late. Strangely, there is no leukocytosis or toxemia. Initially, the patient usually feels well except that he is concerned with his appearance. Not until the downward swing does the patient complain of fatigue. There is no regional lymphadenopathy so characteristic of canceration nor is there the lymphatic spread to satellite lesions seen in fungus infections. The process may be slow or rapid. Disfiguring lesions are markedly manifested during the terminal phase of the disease. The pathologic picture usually consists of a chronic fibrous, nonspecific inflammatory granulation tissue infiltrated by pleomorphic inflammatory cells. There may be one type of malignant nasal granuloma that is associated with necrotizing vascular disease resembling periarteritis nodosa. When this periarteritis component exists, combined clinically with nodular pulmonary granuloma and uremia, it has been termed Wegner's granulomatosis.

Distant granulomatous nodulations (or ulcerations) similar to the original lesion may occur in some cases.[2,3,7] The case presented had thigh ulcerations which initially were nodular, but became umbilicated and then ulcerous. There was also a superficial nodule in the subcutaneous tissue over the supraclavicular fossa which followed the same process. These nodulations may represent consolidation of inflammatory exudates to form the granulomatous nodules.

The midline lethal granuloma is notoriously refractory to all types of treatment. The prognosis is almost universally fatal. For a period of months the case in this report showed arrest and complete reversal of the granulomatous process on large doses of prednisolone. After cessation of the drug and later resumption, the process became completely refractory to all treatment.

Report of Case

The patient, a white woman aged 63, was first seen in November, 1956, complaining of an ulcer that would not heal in her left nasal vestibule. (Two months prior she had consulted her family doctor for a persistent cold and rhinorrhea.) The head and neck examination was completely negative except for a mild granulomatous ulceration of the left aspect of the cartilaginous septum. Multiple biopsies were taken of the suspicious areas, and an absence of bleeding was noted. The tissue was reported as a nonspecific granulomatous process. X-ray of the chest was negative. X-ray of

Fig. 1.—Marked edema and crusting of nose and nasolabial fold and closure of eye, following external skin biopsy (prednisolone started).

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Chestnut Hill Hospital and Hahnemann Medical College and Hospital (Dr. Von Fraenkel). Chestnut Hill Hospital and Jefferson Medical College and Hospital (Dr. Hitschler).

Figure 2

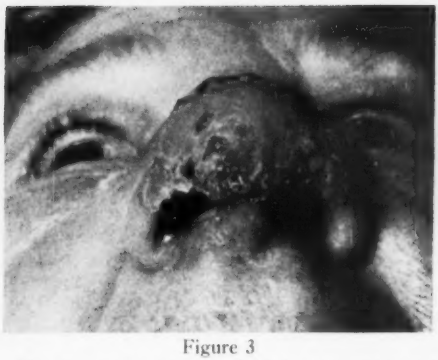

Figure 3

Fig. 2.—Acute edema subsided; nose thickened and nodular. Note involvement of inner canthus.

Fig. 3.—Crusts separating; some induration of columella.

the skull and sinuses demonstrated smooth thickening of the sinus mucosa, which was more marked in the maxillary sinuses. Blood studies, serology, cell counts, and cultures of the nasal scrapings contributed nothing.

Two months later the patient was referred to a dermatologist because of a thickening of skin over the nasal root. A biopsy of this tissue proved nonspecific. The biopsied area refused to heal, and solid edematous induration occurred, which extended in butterfly fashion to both cheeks and closed the one eye. Small ulcers were noted in the posterior pharynx, epiglottic tip, and hard and soft palates. There was further devitalization in the nasal cavity. Serial biopsy specimens, taken from the nasal cavity, palate, and pharynx and sent to various cancer-minded institutions, revealed the same picture of nonspecific granulomatous process.

Up to this point intensive treatment with antibiotics (as per sensitivity tests), heavy metals, and various ointments showed only slight gains. The patient at no time felt ill but was concerned about her appearance and had transient periods of "tiredness."

Rare cures have been reported with cortisone and corticotropin.[7] In March, 1957, treatment with prednisolone was begun, using 60 mg. daily, combined with broad-spectrum antibiotics. At the end of one month the patient exhibited an objective gain. Serial photographs taken demonstrated the progressive improvement. Further, the granulomatous tissue deep within the nose, palate, epiglottis, and oropharynx resolved. The patient was maintained on prednisolone, and the dosage was gradually decreased.

Unfortunately in July the patient began having ulcer-like pains, and a duodenal ulcer was demonstrated on x-ray. The patient was referred to her internist for ulcer management and was seen only at monthly intervals.

In December the patient was admitted to the hospital for intensive studies. She had been taken off prednisolone for the preceding three months because of ulcer management. The lesion across the nasal bridge had extended in a butterfly fashion, and there was marked periorbital and upper lip edema. The granulomatous appearance of her nasal cavity, palate, and oropharynx had recurred. Additionally, there were subcutaneous firm cherry-sized nodules noted in the inner surfaces of the left thigh, calf, and knee, the lateral and medial surfaces of the right thigh, over the right elbow, and in the supraclavicular fossa. The biopsy study reported "masses of cells with irregular shaped nuclei surrounding the blood vessels present. This picture could represent perivascular involvement." Culture of her nose showed a moderate growth

Fig. 4.—Marked improvement. Tissues soft; nasal root impression caused by spectacle nosepiece. Cortisone decreased because of acute duodenal ulcer.

Fig. 5.—Refractory phase.

of Staphylococcus aureus, Hemophilus influenzae, and Pseudomonas. She had a moderate anemia with an average approximately 10 gm. of hemoglobin and a leukocyte count that varied from 5,000 to 10,000, with a normal partition. X-rays of the sinuses demonstrated moderate thickening of the sinus lining, but no bone destruction. X-rays of the chest were negative. The duodenal ulcer had healed completely. BUN, blood sugar, blood cultures, serum studies, electrophoresis, and special stains on repeat biopsies for fungus and tuberculosis contributed nothing.

The patient was placed on triamcinolone (Aristocort) because of the reputed lessened side-effects. The dosage was increased steadily, but there was no improvement and spread of the lesion continued with much heavy crusting and the underlying tissue becoming infected and ulcerated. There was no reactivation of the duodenal ulcer.

An isolated report in the English literature of an x-ray cure[4,6] (although the English radiotherapist was at first frankly skeptical) stimulated us to try a similar dose of deep x-ray—300 r. The patient, however, went steadily downhill and died in March, 1958.

An autopsy showed lesions of both lungs consisting of multiple small abscess-like formations and multiple cavities associated with severe congestation and focal atelectasis, and Brown atrophy of the heart.

This case is presented because of the continued rarity of this disease and because of the dramatic response of the patient to prednisolone, at least initially. The etiology of the disease remains unknown as to whether it is an unusual tumor, an unusual infection, or a defective immunity mechanism. The diagnosis is still made by exclusion of specific granulomata, by the remorseless progression of the disease, usually refractile to all therapy, and by the nonspecificity of the pathologic findings.[6]

1 Summit Ave. (18).

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Clinical Notes, New Instruments and Techniques

A Simplified Technique for Direct Laryngoscopy in Adults

JULIUS PROHOVNIK, M.D., Chicago

In my experience as an otolaryngologist, it has always been a challenge to obtain perfect exposure of the interior of the larynx in order to do precision surgery on the vocal cords. Many times I would blame inadequate anesthesia, poorly relaxed patient, or a peculiar anatomical configuration of the patient's neck, etc., which contributed to my difficulties. When Dr. Sam Roberts of Kansas City introduced his self-retaining laryngoscope, I immediately made use of this instrument, and, although it was an improvement, it still required external mechanical aids, and the scope itself was quite large and not always suitable in each case. Subsequently Dr. Albert Andrews perfected the chest arm attachment so that one could use it with a set of Jackson laryngoscopes. I purchased this attachment and used it on a number of cases with some difficulties at times. Recently Dr. Robert Lewy introduced a self-retaining laryngoscope using a more powerful gear mechanism. From the above, one can gather that the average infrequent

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From the Department of Otolaryngology, Mount Sinai Hospital of Chicago.

Direct laryngoscopy with the patient in a sitting position.

operator such as I, working in smaller hospitals, has found that he needs these various aids in order to facilitate his work.

One day in conversation, Dr. Laurence Rubenstein, a thoracic surgeon practicing in Chicago, told me that he found that having the patient in a sitting position and standing behind the head of the patient he was able to do bronchoscopies with great ease and with very little discomfort to the patient. This suggested to me that perhaps I should try to do a direct laryngoscopy in a similar manner, and so in the next case that came to me in which this procedure was indicated I did a direct laryngoscopy having the patient sitting on a low stool. Standing behind the patient, I had the patient extend his head backward, resting against the front of my body (as illustrated in photograph), and, using a Haslinger laryngoscope, I had no difficulty whatsoever in introducing the scope down into the laryngeal introitus. Both vocal cords and anterior commissure were beautifully in view, and, practically with no effort on the part of my left hand holding the scope in place, I was able to perform a precision biopsy on a lesion seen on the vocal cord close to the anterior commissure. Later I had occasion to repeat the same procedure on six other patients, of whom one was female and five were male. All patients reacted similarly to the first case. There was complete ease in introducing the scope, with perfect visualization of the vocal cords. In addition, the lesion could be clearly seen. The medication in the above cases was as follows: The adult patient whose weight was less than 150 lb. received pentobarbital (Nembutal) 1½ grains (0.1 gm.) an hour and a half before surgery, morphine sulfate ⅙ grain (10 mg.) with atropine 1/150 grain (0.4 mg.) hypodermically, 30 minutes before surgery. In the operating room, the patient's throat was sprayed with 2% tetracaine (Pontocaine). This was followed by the application of 10% cocaine on a curved metal applicator to the pyriform fossa on both sides. Then, using a Lukens cannula with a 5 cc. syringe and using a laryngeal mirror for visualization, I dropped about 3 to 5 cc. of 5% cocaine solution into the laryngeal introitus. For adults weighing over 150 lb. the premedication consists of pentobarbital 3 grains (0.3 gm.) 1½ hours before surgery, followed by morphine sulfate ¼ grain (15 mg.) and atropine sulfate 1/150 grain (0.4 mg.). The local medication is the same in all cases.

In reviewing some of the literature, Dr. Chevalier Jackson mentions the fact that direct laryngoscopy in adults can be done with the patient in the sitting position. He then refers to the original position described by Dr. Kirsten, in which the patient sits on a chair and Dr. Kirsten stands in front of the patient in introducing the laryngoscope. Subsequently Dr. Mouret described a slight modification of the patient's sitting position. He described the patient sitting astride the chair, facing the back of the chair and leaning forward. His contention was that having the patient leaning forward had a great deal to do with facilitating the introduction of the scope. Dr. Chevalier Jackson describes his method, in which the patient is seated on a low stool, an assistant stands behind the patient restraining the patient's head, and the surgeon sits on a high chair in front of the patient.

I submit my method for my associates to try and judge for themselves as to its merits.

5718 W. North Ave.

A New Procedure for Evacuating Fluid from the Middle Ear in Cases of Secretory Otitis Media

L. Q. PANG, M.D., Honolulu

In the treatment of secretory otitis media in which the secretion is a serous type, one of the best methods of removing secretions from the middle ear after a tympanotomy is by means of the Siegle otoscope or the Bruening-Zeiss otoscope. Even after inflation of the middle ear fails to blow out any more secretion from the middle ear, the use of massive suction by the Siegle otoscope usually succeeds in drawing out secretions from the middle ear.

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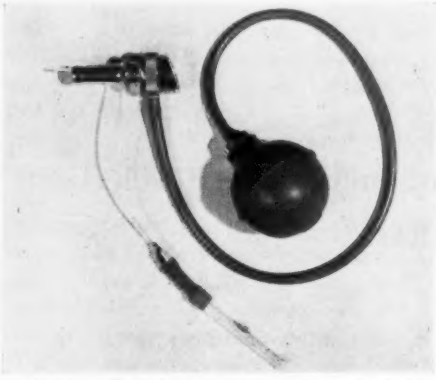
However, it is very frustrating and exasperating to see the solution withdraw back into the middle ear after the suction is released. Some have attempted to overcome this by inserting a small wick of cotton in the external canal during the procedure to absorb the secretion when it comes out from the middle ear through the perforation. The cotton soaks up the fluid as a blotter soaks up ink. This procedure, however, is time-consuming in that the cotton wick has to be changed frequently.

An apparatus has been designed to improve the procedure (Figure). It consists of a Bruening-



Showing Bruening otoscope with polyethylene tube protruding from speculum.

Zeiss otoscope in which a small polyethylene tube has been inserted through a small tight fitted opening in the speculum. The polyethylene tube is connected by a small needle to a rubber tube and thence to a suction apparatus. The other end of the polyethylene tube is then inserted near the perforation, where it siphons off the secretion as it is sucked out of the middle ear by the massive suction created by the Bruening-Zeiss otoscope.

When the suction by the bulb is exhausted, the speculum need not be removed. By squeezing on the bulb, positive pressure is applied after which vacuum is again reestablished by releasing the bulb. This procedure may be repeated until no more fluid is evacuated. The positive pressure serves to aid in the evacuation of the fluid just as the injection of air into a half-empty bottle aids in the evacuation of a solution into a syringe.

This apparatus and procedure have been used in many of our cases and have been very helpful. It is important to emphasize that this procedure does not work in cases where the secretion is of a thick mucus type.

1374 Nuuanu Ave.

Cylindroma of the Trachea

Report of Case

RAHIM SOLTANY-AZAD, M.D., Shiraz, Iran

A 51-year-old white man was admitted to the Nemazee Hospital on June 1, 1957, with the chief complaint of difficulty in breathing.

He was perfectly well until six years ago, when he developed a mild cough that did not respond to any medication. At the onset he did not bring up any sputum, but about two years ago he began to produce some bloody sputum. Eight months ago he gradually developed dyspnea, for which he was finally admitted to the hospital.

Physical examination revealed a well-developed and well-nourished man who was in severe respiratory distress. The blood pressure was 110/70; the pulse was 76; the respiratory rate was 24, and the temperature was 36.9 C (98.4 F). Direct laryngoscopy revealed a lobulated tumor that had a reddish smooth surface and occupied almost 9/10 of the tracheal lumen and was attached to the right anterolateral tracheal walls. The remainder of the physical examination was unremarkable. X-ray showed emphysema of both lungs. Bronchography revealed a rather large soft tissue mass 4-5 cm. below the larynx, extending for at least 5-6 cm. in the right anterolateral portion of the trachea. The lesion had a flat base and involved the tracheal wall. Microscopic examination showed a cylindroma.

An incision was made on the anterior midline of the neck, and the tumor was removed surgically. Ten days after the operation x-ray therapy was started, and within 49 days a total dose of 3,000 r was delivered.

He was discharged on Aug. 15, 1957, in good condition. Bronchography at that time did not show any residual tumor tissue. No recurrence has been reported up to the present.

Nemazee Hospital.

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Eye, Ear, Nose, and Throat Department, Nemazee Hospital.

Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear

Summaries of the Bibliographic Material Available for 1954

BRUCE PROCTOR, M.D., Detroit; EMERY PICK, M.D., Los Angeles; MICHEL PORTMANN, M.D., Bordeaux, France, and RICHARD BELLUCCI, M.D., New York

(Concluded from page 371)

The Ear in Head Trauma

After skull trauma, according to Piquet and Decroix,[217] it is possible to find many varying clinical characteristics of deafness—in short, the most frequent nature of hearing loss is a perception deafness, but we also see the mixed type. Pure transmission or total hearing loss are exceptional. Frequently the hearing recovers during the first or second month after the trauma, but in some cases the defect persists. We can consider it as permanent when it is present for six months. The hearing tests often show that recruitment is at first negative, then positive. The authors think that it is the result of vascular phenomena which appear in the circulation of the inner ear and of the cochlear nerve and which are due to the fragility provoked by the trauma.

From anamnestical data of 1,188 cases with craniocerebral injuries, damage to the cochlear nerve was established by Djordjevic and Perisic[69] in 11.4% and to the vestibular nerve in 40%. There were mostly fractures of the temporal bone. However, certain cases showed labyrinthine lesions without any objective pathological finding in the tympanic membrane or the pyramid. These lesions are interpreted as results of commotio labyrinthi. The patients should remain under medical observation for at least one year in order that the restoration of the eighth nerve may be controlled. Only then may a judgment of the patient's condition be established.

In cases of craniocerebral trauma, an auditory scotoma is known to exist around the frequency of 4,096. Five cases are reported by De Reynier,[61] where, between 4,000 and 6,000, the audiogram showed that the curve of bone conduction was less affected than that of air conduction. In general, this is characteristic for lesions of the transmission apparatus. If, however, the results of acoumetry and audiometry are compared, it is seen that the transmission apparatus is little affected (positive results of Rinne's test, no elevation of the inferior threshold). It would seem probable that the trauma, which, to provoke a perception deafness, would attack Corti's subepithelial system, would have struck the epithelial system instead and thus have caused a transmission deafness indirectly, via the ossicular muscles. If there exists a dependence of the middle ear upon the internal ear, it appears that the reverse relationship is not excluded. The author admits that commotion of the labyrinth, a primary lesion of the internal ear, leads indirectly to contraction of the ossicular

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Associate Professor of Otolaryngology, Wayne State University (Dr. Proctor); Associate Professor of Otolaryngology, College of Medical Evangelists (Dr. Pick); Professor Agregi à la Faculté de Médicine, University of Bordeaux (Dr. Portmann); Associate Professor of Otolaryngology, University of New York (Dr. Bellucci).

muscles, with predominance, of bone conduction especially, for frequencies of mean value (4,000-6,000), which induce the strongest contractions.

Study was made by Anderson [7] of the variations of optokinetic nystagmus in 154 cases of cranial trauma and cerebral concussion. In cases of slight commotio cerebri and without other neurological complications, optokinetic nystagmus never showed appreciable alterations, whereas in 4 of 54 cases of severe cranial trauma with cranial fractures and concussion of prolonged duration, no optokinetic nystagmus could be demonstrated. In conclusion, it is stated that in patients with trauma to the skull, the absence of optokinetic nystagmus is of value for focal localization, but only when it is accompanied by hemanopsia or other clinical symptoms, which render the hypothesis of a lesion in the classical localization probable. In the other cases the interpretation of the absence of optokinetic nystagmus is doubtful. From the point of view of forensic medicine, the unilateral absence of optokinetic nystagmus is a valuable symptom as it renders certain the diagnosis of a lesion in one cerebral hemisphere. The optokinetic postnystagmus, also present in perfectly normal persons, is of no value.

Tinnitus

The scala media and the hair cells are believed to provide an electric current which is varied in strength by the movements of the hairs of the hair cells, reports Davis.[55] The variations in current are the basis of the cochlear microphonic (the Wever and Bray) effect. This alternating current excites nerve impulses, but the steady current flow also may be strong enough to excite a slow "spontaneous" discharge which would be the basis of tinnitus. One way in which the balance can be upset in the latter direction is the mild irritation which is produced by exposure to very loud sound. A steady mechanical pressure that causes a slight permanent displacement of the tectorial membrane in the proper direction relative to the hair cells could also be expected to cause tinnitus. There is no particular reason, on this theory, to expect tinnitus as a result of anoxia, and the author believes that theories relating tinnitus to vascular spasm of the stria vascularis are probably on the wrong track. He believes that the basis of tinnitus is to be sought in disturbances of the postulated regulatory mechanism for the excitability of the sense organ.

Tinnitus is classified by Ruis and Griot [245] as follows: (1) tinnitus due to alteration of or interference with the transmission mechanism of vibratory energy (either by air or by bone conduction) to the receptor; (2) tinnitus due to alteration of the organ of Corti, where vibratory energy is transformed into bioelectric energy; (3) tinnitus due to alterations of the central pathways in the first, second, third, or fourth neurons; (4) tinnitus due to alteration of the conscious perception. Examples of each of the above-mentioned groups are given through the description of clinical records of typical cases.

Goodhill [112] makes the following subdivision of "true" tinnitus: (*a*) tinnitus caused by the removal of the normal masking effect of surrounding ambient noise (otosclerosis, chronic otitis, glomus jugulare tumor, etc.); (*b*) neural discharge tinnitus, i. e., a suprathreshold auditory paresthesia (traumatic deafness, hydrops labyrinthi, etc.). Further on the tinnitus may be compensated or decompensated. Here the psychological circumstances are of importance. Therapy may be otological, palliative, the use of ambient noise, sedation, psychotherapy.

Mizuochi [200] states that the pitch of tinnitus aurium at its maximum may never measure more than 10 db. Consequently, the actual impairment of hearing that the presence of tinnitus may cause, if any, should be slight. The author considers any form of tinnitus to be a paresthesia.

The tinnitus reported by Versteegh [280] under discussion was caused by rhythmic contractions of the muscles of the soft palate (50 times per minute). It disap-

peared when the patient was under anesthesia. Narcoanalysis and after-treatment by suggestion led to recovery.

Nicotinic acid was used intravenously and subsequently perorally by Flottorp and Wille[91] to treat 22 cases of tinnitus. The patients were thoroughly examined before, during, and after the injections, both clinically and audiologically (tinnitus pitch, loudness, masking). The cases were grouped with respect to audiological findings and etiology. Some of these groups (high-frequency, cryptogenetic and Ménièriform tinnitus) seem to have reacted favorably to the treatment. There is good agreement between the subjective statements of improvement by the patient and the audiological measurements. Nicotinic acid (1%) was injected intravenously in daily increasing doses from 25 to 100 mg. with an increase of 5 mg. daily. The injections are performed slowly in the course of a few minutes, and the patient rests for 15 minutes after the injections. Continued medication with nicotinic acid is performed with two tablets (0.1 gm.) twice daily before meals.

Atkinson[15] reports that in 10 patients with intractable tinnitus and hearing loss (excluding otosclerosis) already under intensive therapy with vitamin B, the addition of vitamin A parenterally and orally was without effect.

Malingering

To prove malingering in hearing loss cases in the absence of an admission is most difficult with our present testing methods, states Glorig.[109] The relationship between pure-tone and speech audiometry test is effective in disclosing a functional hearing loss because a speech signal is a transient sound and therefore is difficult to match in loudness against an established spurious pure-tone loudness level. This results in a discrepancy, usually over 12 db., when the malingerer is tested by pure tone and speech. The Doerfler-Stewart test is designed to break down the established spurious loudness level by superimposing a specially designed noise over a speech signal. This test should be routine in cases of malingering. The psychogalvanic test, eye blink, and electroencephalograph test lower order neural mechanisms, and, if positive, indicate neural activity which includes the auditory system from the standpoint of continuity of transmission of a sound stimulus. These tests cannot differentiate between a conscious and a subconscious block. Delayed speech feedback offers a method which will differentiate malingering from psychogenic overlay. The malingerer can hear and admits it to himself but not to others, but the psychogenic does not admit hearing even to himself. If the delayed speech feedback circuit test is applied to a case with a psychogenic overlay, the delayed speech should not confuse him, but the malingerer should be confused because he is unable to control the effects of the delayed speech on his monitoring feedback circuit.

Psychogenic deafness is classified by King[158] as follows: 1. Functional deafness—(*a*) loss of discrimination faculty; (*b*) organic deafness with hysterical overlay; (*c*) pure hysterical deafness with no evidence of organic defect. 2. Malingering—(*a*) malingering associated with hysteria; (*b*) malingering with organic defect; (*c*) simple malingering. Loss of the capacity for discriminative listening arises from a voluntary or involuntary exclusion of sound by the patient, generally found in older patients but also in young children with marked hearing loss. A history of sudden onset of deafness should be viewed with suspicion; likewise, if the history of deafness does not coincide with the normal clinical history in assessing the deafness from the organic standpoint, hysteria may be suspected. 3. Psychogenic loss may be suspected—(*a*) if speech reception is inhibited by a noise interference level less intense than that at which speech was presented; (*b*) where noise perception threshold is lower than that for speech reception; (*c*) where there is a marked discrepancy between speech thresholds. Successful management depends on early diagnosis. Dilute

intravenous thiopental (Pentothal) sodium is given, and suggestive therapy is applied in the period of recovery. Simulated deafness may be associated with organic deafness and sometimes with hysteria. Generally only unilateral deafness will be simulated. Detection is with Stenger's Lombard's test and audiometry with variable patterns of testing.

Pseudo-organic deafness in children is discussed by Getz.[106] It resembles organic deafness because of the child's hearing difficulty in interpersonal situations and a consistent audiometric indication of loss resembling the conduction or perception loss curves. If a broader examination is done, including voice and speech analysis, simplified word perception test, and a history of intrafamily dynamics, the diagnosis of organic loss must be abandoned. Psychological therapy, rather than a mechanical solution, is indicated. The disorder is best understood as the mechanism whereby the person excludes, at convenient times, verbal communication.

A successful test of simulation deafness or psychogenic deafness is that it be simple of administration, capable of interpretation by a reasonably intelligent layman, and productive of reliable, repeatable results. It should also result in some reasonable approximation of the "true threshold," especially in cases of partial psychogenic overlay. Such a test involving the use of the delayed side-tone phenomenon is reported by Hanley and Tiffany.[125] The subject is merely asked to read a simple passage while he wears a pair of earphones. As he reads, his voice is recorded and then, with about a 0.2 second delay, is fed back at a controlled intensity level to the subject's earphones. An immediate and profound change is observed in the speech of the hearing subject. His enunciation is slurred; he stutters; changes occur in voice quality; pitch control is lost; intensity is increased, and the speech is markedly slowed. The effect occurs reliably at from 20-40 db. above threshold. Whistling seems an ideal activity, not only because of rhythm and pitch control required but also because bone-conduction sound is minimal compared with voiced speech. Even if motivation for malingering seems clear-cut, it is probably wise to seek psychiatric council, not only for aid in interpreting the meaning of the examination findings to a possibly disturbed person but also to aid in establishing a differential diagnosis between malingering and hysteria.

Skillful malingerers may produce very similar audiograms at various intervals, reports Hager.[120] The memory of differences in tone intensity is sometimes astonishing. On the other hand, somewhat different audiograms when repeated are not always a sign of malingering. The technique of measuring is very important. But always turning the audiometer controls at the same speed is a frequent mistake and helps the malingerer. In suspected cases the examiner has to constantly modify his audiometric speed and procedure.

A test for the recognition of unilaterally simulated deafness is reported by Matzker.[186] The test is based on two psychoacoustic principles: 1. During binaural presentation of two tones of equal frequency only the louder tone can be heard (Stenger test). 2. Whenever the amplitudes of two tones of almost equal frequency become identical, the audible interferences (beats) show the additional phenomenon of tactile beating or throbbing. By means of an audiometer and tuning fork (of almost equal pitch) the author demonstrated a new test which uses these physiological principles. It appears to be equally reliable in cases of hypacousis. A malingerer, however, would fail to give the unexpected physiological responses.

The "retarded voice" method proposed by Azzi was applied for the detection of deafness malingerers by Tenaglia.[271] The method is based on the deafening of the ears caused by the retarded voice of the patient when the latter speaks or reads aloud. The apparatus consists in an ordinary tape recorder with double reproduction at an interval. The method was applied to

tests given to military personnel when the usual audiometric tests had left doubt as to the reality of their deafness. From the results obtained it is concluded that the test is of considerable interest, as it is objective and sufficiently sensitive.

Ménière's Disease

The theory that Ménière's disease is due to rupturing of subepithelial vesicles on the walls of the semicircular canals and contamination of the endolymph by this vesicular fluid, as proposed by Lempert et al., is refuted by Seymour,[258] who found these vesicles in all normal and pathological temporal bone specimens that he examined. These vesicles appear to be of doubtful pathological significance. The preponderance of evidence instead points to a vascular cause for Ménière's disease.

The vesiculated epithelial excrescences described by Lempert have been noted and described by many earlier investigators, first by Rudinger, in 1864. These excrescences can be found in nearly every normal labyrinth. Wustrow[299] believes that these excrescences are subepithelial. The subepithelial layer has fine, filiform partitions dividing it into small sections and compartments; some of them form the vesicles.

Ménière's disease, serous labyrinthitis, and stagnation hydrops of the inner ear are exudative diseases of the inner ear, according to Brunner.[35] In stagnation hydrops the effusion is caused by an impediment to the venous outflow (tumor in posterior fossa). Serous labyrinthitis is due to inflammation in the tympanum or meninges. Ménière's disease develops if there is a constitutional tendency to vasospasm, which is perhaps identical with physical allergy. When the constitutional tendency becomes activated, it results in a spastic atonic state of the blood vessels of the inner ear. The spasm involves the capillaries and venules. Ménière's disease is frequently associated with involvement of the cisterna pontis and the patient complains of postauricular or suboccipital headaches. In these cases Brunner suggests draining the cisterna pontis and dividing the ductus endolymphaticus.

A review of 900 cases of Ménière's disease is presented by Cawthorne and Hewlett.[41] In 13% both ears were affected, half of these since the onset of the disorder. Most commonly deafness and vertigo developed simultaneously. Allergic disorders were found in less than 5%. Distortion of hearing is considered often to be a valuable distinguishing feature of endolymphatic hydrops; it is demonstrated by contrasting the relatively poor speech audiogram with the relatively good pure-tone audiogram. Distortion of hearing was noted in half the cases. The vestibulotoxic properties of streptomycin may be used to destroy the labyrinth, but should be limited to young people with bilateral disease, since older people are too incapacitated by bilateral vestibular destruction. Nerve section should be used only when the hearing on the affected side is remarkably good. Current views on pathology and treatment are discussed with reference to favorable results obtained in 288 unilateral cases submitted to membranous labyrinthectomy.

The peripheral vestibular organ is controlled by the cerebral cortex, which was proved, on the one hand, by experiments with decortication; on the other hand, by the training of conditioned reflexes after vestibular stimulation. In everyday life, vestibular stimuli over the threshold are frequent, but, if they are not too strong, they are suppressed by inhibitory centers of the cerebral cortex. It must be assumed that in Ménière's disease the ability of suppression is eliminated. Disturbances of other analyzers also affect the vestibular stimuli, predominantly through conditioned reflexes. Directives for the prevention, rehabilitation, and treatment of Ménière's disease, which are based on these theoretical explanations, are given by Chladek.[47]

Fifty cases of Ménière's disease were examined audiometrically by Hahn[122] and with all three vestibular tests. The main findings were as follows: recruitment and elevated galvanic threshold (10 cases); no

response (32 cases); recruitment and elevated galvanic threshold (6 cases); negative recruitment and normal galvanic response (2 cases). Similar to the negative recruitment, an elevated galvanic threshold may indicate a retrolabyrinthine seat of the lesion.

A study was made by Bonati [26] of microscopic alterations of the labyrinth in rabbits 10, 20, 30, or 50 days after alcoholization (Mollison) or diathermocoagulation (Day) of the posterior labyrinth. It seems that alcoholization reproduces not only the lesser injuries, consisting of hemorrhage, serofibrinous inflammation, forming of granulations in the semicircular canals, but also constant alterations in the cochlea, such as regressive processes in the spiral ganglion, amorphous exudate, and distention of the basal membrane. The alterations after electrocoagulation are of the same order but of a more serious character. An intact cochlea was found in only a few cases.

For the purpose of determination of injury compensation in Ménière's syndrome, Zatloukal [304] employs Vojacek's otolith reaction for the evaluation of subjective disturbances. He divides subjective complaints into two categories of giddiness: (1) tendency to vertigo—temporary or persistent; (2) real vertigo—temporary or persistent—accompanied by falling and vomiting. He estimates patients suffering from giddiness at 0-20% and from real vertigo at 10%-60% of full injury compensation. Functional alterations after Vojacek's otolith test of zero and first degree correspond to 0-10%, those of second degree to 20%-40%, and those of third degree to 40%-60% of full injury compensation. In the administration of invalidity compensation a certain degree of vestibular alteration was taken into consideration with regard to original profession of the patient.

An attempt was made by Derlacki [62] to evaluate the results of the small optimum dosage of histamine in the treatment of 55 cases of Ménière's disease. The optimum dosage technique begins with 0.1 cc. of a 1:100,000,000 dilution of histamine diphosphate solution given subcutaneously. If no noted effect follows within 24 hours, then the dosage is increased to 0.1 cc. of a 1:10,000,000 dilution given one to three days after the first injection. The dosage is increased until the optional dosage or that amount which will just relieve the symptoms is reached. Once the dilution has been found which will give a day or so of improvement, the dosage is increased from 0.1 cc. to 0.25 cc., to 0.50 cc., etc., of that dilution. Best results are on a subcutaneous dosage which maintains the patients symptom-free for intervals of three to seven days, supplemented by sublingual administration of histamine one or two dilutions stronger than the subcutaneous dosage, using 2 drops twice daily. When vertigo ceases and hearing has been stabilized for six to eight weeks, the histamine is discontinued. Recurrence calls for resumption of the complete program.

In the treatment of edema in any organ, be it the brain, the heart, or the labyrinth next to "antiretentional" diet, diuresis is the most important therapy. Foldes [92] administered aminophylline tablets, 0.1 gm t. i. d. and meralluride (Mercuhydrin) 1 cc. intramuscularly, once weekly in cases of Ménière's disease. Remission lasted for many months. As in the treatment of migraine and angina pectoris, resumption of active therapy was followed by renewed response.

Foxen [98] reports the use of streptomycin in Ménière's disease. Before this method is used all other more conservative methods of treatment should have been used and the affliction should be bilateral. Before treatment is started renal function should be carefully assessed, since toxic effects seem to occur more rapidly when it is impaired. The age of the patient must be considered since the process of adjustment to an "avestibular life" is very slow in the elderly. During the treatment cochlear and vestibular function should be checked regularly. Hearing tests are done at two-day intervals. Any loss of hearing requires immediate withdrawal of the drug. The role of the

physiotherapist in rehabilitation is very important.

Among the methods for destruction of the labyrinth, the technique of Lempert, Day, and Cawthorne is preferred by Brown.[32] The method of Dandy with complete resection of N-8 is considered theoretically the most satisfactory because the most serious cases of tinnitus are best relieved in this way. But a follow-up of Dandy's cases proved that in complete section of the acoustic and vestibular nerve 9% of the patients had a complete facial paralysis. For this reason this method cannot be recommended in all cases.

A report is presented by Arslan and Baccaglini [12] of six cases of Ménière's disease subjected to antrotomy for identification of the lateral semicircular canal and then treated with direct application of ultrasonics to this canal for periods varying between 8 and 15 minutes. The purpose of this treatment was to suppress the vertiginous attacks by destroying the ampullary receptor by means of the ultrasonic waves. The technique of the combined surgical and ultrasonic intervention is described, and a report is given of the results. These were quite encouraging; the attacks of vertigo disappeared and improvement of the auditory acuity and tinnitus was seen to follow in all cases. The mechanism of the action of the ultrasonic waves on the labyrinthosis is discussed.

Flett,[90] in analyzing the results of the saccus operation in 74 cases of Ménière's disease, concluded that this operation offers a 25% chance of retaining the hearing and stopping the tinnitus and giddiness, enables 61% to continue at work without giddiness but with gradual loss of hearing, and fails in 14%. Flett considers it worth while to continue this saccus operation as a preliminary to doing the destructive operation since need for destructive surgery may be obviated.

The histological findings in the temporal bone of a case of Ménière's disease treated by Portmann's operation are reported by Hallpike and Harrison.[124] The patient died on the third postoperative day. In the affected cochlea Corti's organ was obviously shrunken with disorganization of the outer hair-cell complex. Reissner's membrane was found to be still bulging in spite of decompression of the saccus at operation. This does not mean that in the autopsy there was a raised pressure forcing the membrane into its new position. The purpose of Portmann's operation would still be fulfilled since further outpouring of endolymph would be discharged through the open saccus and there would be no further displacement of Reissner's membrane.

A modification of Portmann's operation for Ménière's disease is described by Yamakawa and Naito.[300] Portmann's operation consists in incising the lateral wall of the endolymphatic sac, i. e., an extradural incision. It is claimed that some of the failures, when this technique is used, may be due to the operative area filling up with granulation tissue. In order to eliminate this hazard, a technique has been developed in which a window is made in the medial wall of the endolymphatic sac. This entails incising the dura to obtain access to the medial wall of the sac. This method enables endolymph to flow constantly into the spinal fluid. Twelve cases have been subjected to this operation. Vertigo disappeared in eight and was improved in two. Tinnitus disappeared in one case, was diminished in seven, and its character changed in one. Hearing improved in five cases, was unchanged in two and deteriorated in four.

Patients with Ménière's disease who have had a fair trial on medical treatment but who persist in having severe attacks of dizziness with loss of useful hearing in the ear involved, with fair or good hearing in the opposite ear should have surgery for relief. Under local anesthesia Goodyear [115] opens the mastoid cortex with a Hudson brace and bit and follows with the Adson burr. The exposed horizontal canal is opened anteriorly with curettes. Through this opening is passed a small dental explorer through the ampulla and into the

vestibule. In 22 cases complete loss of hearing and vestibular function occurred.

According to Johnson,[148] acute fulminating Ménière's disease or acute perceptive deafness should be handled as an acute surgical emergency to prevent permanent irreversible deafness by procaine stellate ganglion block. If two or three attacks are controlled satisfactorily, a preganglionic dorsal sympathectomy is advised. A similar program of treatment is carried out for tinnitus and some cases of nerve deafness. A psychosomatic overlay frequently occurs in Ménière's disease and may be the result of afferent impulses arising from the troubled area of the cerebral cortex and passing to the hypothalamic center, disrupting autonomic equilibrium.

The effects of cervical sympathectomy in 18 cases of Ménière's disease are reported by Lewis.[173] Stellate ganglionectomy resulted in considerable improvement in seven, slight improvement in four, doubtful results in two, and failures in four with regard to vertigo. Audiometry failed to show convincing improvement in hearing in any case. Intolerance of loud sounds improved in 6 of 12 cases. As to tinnitus, five improved, six improved but relapsed, and seven were unchanged. Tinnitus had not been abolished in any case. Nine cases with distortion improved in three. The operation seems most likely to succeed in the young and early middle-aged patients without profound deafness. The more recent the onset of vertigo the better the prognosis. Where one ear is completely or severely deafened as the result of Ménière's disease or labyrinthectomy and the better ear becomes the site of active Ménière's disease, cervical sympathectomy should be considered. If Ménière's disease does not respond quickly to medical measures, the operation should be done before irreversible changes occur in the inner ear. The value of stellate ganglion injection in selecting cases for operation is doubtful.

The surgical techniques usually applied in the treatment of Ménière's disease are reviewed by Menzio [193] and a description is given of a personal method which consists in functional suppression of the ampullar crest of the lateral semicircular canal without opening the membranous labyrinth. This can be achieved by introduction of fibrin sponge into the perilymphatic space of the lateral semicircular canal, which causes compression and obliteration of the endolymphatic space. The canal is opened via the transmastoid approach, without damage to the tympanum or to the ossicles. This technique was applied in 12 patients; the postoperative course was fairly uneventful. Nystagmus lasted only for four or five days, and the patients could leave their beds as early as the fifth or sixth day. Control tests by means of vestibular stimulation (galvanic, thermal, rotatory) revealed complete suppression of the lateral semicircular canal. A few months after the operation, some of the patients reported total disappearance of the vertiginous symptoms.

In 100 patients Rosen [233] exposed the chorda tympani, using an endaural inferior circular incision in the external meatus, about 6-7 mm. lateral from the drum. Electrical stimulation of the chorda tympani caused vertigo (4 cases), vertigo and tinnitus (8 cases), tinnitus alone (79 cases), pain (80 cases), facial contraction (82 cases), and taste sensation in none of the cases. In 97 cases, section of the chorda was performed in treatment of Ménière's disease; 49 patients obtained complete relief, and 21 little or no relief. The rationale for the operation is the assumption that there exist central connections between the central afferent nucleus of the chorda and the vestibular nuclei. The operation should be taken into consideration before destruction of the labyrinth is contemplated.

According to Rosen,[234] stimuli along the chorda tympani nerve may in some cases be responsible for Ménière's disease. On that basis he advocates section of the chorda tympani. In 41 out of 50 cases he obtained excellent results; tinnitus also ceased in half of them. Rosen had observed that when the chorda was sectioned during fenestration operation, dizziness was consider-

ably reduced. As the anatomic basis underlying the effect on vertigo, one would have to assume connecting branches between the nervus intermedius and the vestibular nerve; with regard to tinnitus there may be connections between the tractus solitarius area and that of the nuclear area of the cochlear nerve. Occasional recurrences are believed to be on a functional basis.

Vertigo in five cases following radical mastoidectomy with sacrifice of the chorda tympani nerve was relieved by section of the tympanic plexus, reports Rosen.[235] He suggests the hypothesis that the tympanic plexus, as well as the chorda tympani, carries afferent impulses to a sensory nucleus (presumably the nucleus of the seventh nerve) which has functional interconnection with the vestibular nucleus. The abnormal activity of these afferents causes the vertigo.

Vertigo

Jordan[150] remarks that much confusion has arisen from the different interpretations of Ménière's disease. There is a tendency to call all vertigo-deafness-tinnitus combinations Ménière's disease. The vertigo in Ménière's disease is severe, explosive, and may last from a few minutes to hours; nausea and vomiting are far less bothersome. Vertigo may strike daily or at six month intervals. The deafness is usually unilateral, of cochlear type, uniform in all frequencies, and fluctuating. Distortion, diplacusis, and sensitivity to loud sound are frequent; pressure feeling may be an early sign. Tinnitus is of the low, roaring type. The caloric response is usually depressed. The hearing disturbances are definitely in the foreground, and in one-third of the cases they are a much earlier complaint than vertigo; on the average there is a three and one-half year time lag. In the arteriosclerotic type of Ménière's syndrome, the hearing loss is bilateral, with more pronounced high-tone loss. There is no fluctuation, no distortion, and tinnitus is high-pitched. Autonomic dysfunctions are more frequent in women, mostly around 40, particularly in allergic patients under emotional stress and tension. The hearing difficulties are minor. Vertigo caused by intoxication or virus infections is not accompanied by loss of hearing. On the other hand, there is a total loss of hearing when hemorrhage into the labyrinth occurs. The medical therapy of the Ménière syndrome has three basic forms: (1) dehydration; (2) vasodilatation (nicotinic acid); (3) desensitization. The author advocates small doses of histamine.

The anatomical and etiological diagnosis of vertigo is based not only on a good vestibular examination but also on cochlear tests. Chavany et al.[43] analyzed the principle of the vestibular diagnosis: (*a*) The vestibular reflexes are of peripheral type, total and harmonious. This is a purely otological disease like Ménière's disease, vertigo of otitis, the progressive deafness, the hearing loss due to certain traumas. (*b*) The vestibular reflexes may be of peripheral type but with atypical signs, so a radicular syndrome is pointed out. (*c*) The vestibular reflexes may be disharmonious; then they constitute a central syndrome included in a neurological picture. (*d*) Finally, the results are not always too precise. Certain patients complain of vertigo, but the vestibular tests are normal and the etiology is not well defined, so frequently we must investigate the field of general medicine to explain this vertigo.

Kunkle[163] presents the view of the neurologist concerning central causes of vertigo. The first step is to determine that the symptom is true vertigo. The central pathways concerned are described. Some diagnostic features are helpful in the differentiation between central and labyrinthine: The sensation is usually rotary; the timing episodic; tinnitus and deafness are common; nystagmus is fine and fast; caloric responses of all varieties and other neurologic abnormalities do not exist. In central vertigo the sensation is more variable; the timing is sustained; tinnitus and deafness are uncommon, nystagmus coarse and slow,

caloric responses are of all varieties except hyperactive, and other neurologic abnormalities are common. Central vertigo may be due to a pontine lesion, mostly due to (1) vascular changes; (2) rhombencephalitis; (3) tumor of the brain stem; (4) multiple sclerosis. Central vertigo may also be due to parapontine lesions and other cerebral disorders, like the aura of migraine, and epileptic seizure. Vertigo in post-traumatic states is temporary, even if it should last for many months. Vertigo as a reflex from neck disease is somewhat obscure, yet it perhaps explains best the post-traumatic vertiginous disorders. Vertigo caused by vestibular neuronitis or viral infections is never accompanied by hearing loss and tinnitus; occasionally the caloric response may be depressed. In emotional patients a psychogenic etiology should always be taken into consideration.

Postinfectious meningoencephalitis as an etiologic factor in certain cases of vertigo is discussed by Herberts.[129] Centrally released vestibular symptoms are frequently induced by encephalitis processes. In benign cases there is no inflammation of the brain tissue; the term encephalopathy is more adequate. Experimental and clinical results indicate that in cases with serious general infection a plasma leakage takes place in the brain as a result of a disturbed function of the vessel endothelium. This causes toxic or neuroallergic lesions of the nerve cells, reversible or persistent (polioencephalopathy). The etiologic factor in most cases is a virus infection.

Schröder[254] reports the case of a 51-year-old woman with bilateral inner ear deafness of three years' standing and tinnitus of shorter duration. The history revealed sciatica and rheumatism in both arms. Considerable narrowing of the intervertebral foramina at C-6 and 7 was demonstrated, and the patient was referred for orthopedic treatment. After the sixth application of extension to the vertebral column by means of the Glisson sling, the patient experienced vehement vertigo, whereupon tinnitus ceased and hearing improved until it was practically normal. This case shows the principal symptoms of Ménière's disease, but in reversed sequence. It differs from the cases described by Lermoyez in that he observed spontaneous improvement of the hearing and vertigo, but the pathologic basis is the same: osteochondrosis of the cervical spine. On the other hand, the condition differs from Ménière's disease (endolymphatic hydrops). Lermoyez's syndrome is not as rare as had been assumed, yet in 25 cases of roentgenologically demonstrated osteochondrosis at the level of C-5 and 7, vertigo occurred only in two instances, deafness traceable to the changes in the cervical spine only once. The connection between cervical osteochondrosis and inner ear symptoms is not of a mechanical nature, but due to stimulation of the sympathetic nerve. Consequently the term "syndrome of the cervical sympaticus" is proposed.

Studies were carried out by Hahn[121] in two groups of patients with labyrinthine vascular disease: (1) subjects with a symptomatology classifiable as Ménière's syndrome due to endolymphatic hydrops, which group comprises only those patients who suffered from objective attacks of vertigo of long duration, accompanied by nausea and vomiting and associated with hypacusia and tinnitus aurium; (2) subjects affected with labyrinthine angioneurosis, which group contains the patients with subjective vertigo of brief duration with little or no cochlear involvement. From the findings obtained, it appears that both in endolymphatic hydrops and in labyrinthine angioneurosis there is a predominance of subjects who are markedly sensitive to histamine over those sensitive to nicotinic acid. The latter category was much better represented, however, among the group with labyrinthine angioneurosis than in the endolymphatic hydrops group. A consideration of the variations of the tests in the subjects affected with endolymphatic hydrops in relationship with the development and the clinical type of the cochleovestibular symptomatology shows that the tests with histamine and with nicotinic acid are positive

in the greater number of subjects affected with initial Ménière's syndrome or due to endolymphatic hydrops with cochleovestibular involvement. As a rule, both tests gave a negative result in patients suffering from Ménière's syndrome with neuritic evolution. It was not possible to confirm that the results of the intradermal histamine test may be associated with certain characteristics of cochlear vertiginous disturbances.

Description of a syndrome composed of extremely diverse symptoms and signs and often mistaken for neurasthenia or neurosis is reported by Gayral and Neuwirth.[103] The cardinal symptoms and signs are sympathicogenic headaches and facial pains, and pains in the back of the neck; vasomotor disturbances, particularly affecting the face, the ears, and the oropharynx; vertigo, tinnitus, asthenia, insomnia, and photophobia. The retinal arterial pressure is generally decreased. Ocular lesions and paroxysms of orbital pain may occur. Diagnosis is based upon careful and judicious questioning and thorough physical survey and x-ray exploration of the cervical segment of the vertebral column.

Indications and results of infiltration of the vertebral nerve in dizziness of cervical origin are presented by Gignoux, Takizawa, and Bonnefoy.[107] The dizziness is brought about by vertebral arthrosis or cranial trauma. On clinical examination, various forms with predominant influence of the vasomotor nerves are distinguished. These are better influenced by way of the vertebral nerve than by way of the carotid nerve. A technique of infiltration of the transverse canal by the anterior route is described. The method is simple. The risks and the results are described.

Maspetiol et al.[181] describe the "posterior cervical sympathetic syndrome" of Barré and then propose a treatment of the vertigo due to this disease. This syndrome in several cases occurs in a type which predominates in vestibular symptoms, more or less connected with cochlear symptoms. A cervical origin must be suspected if the vertigo occurs only when a certain position of the head is adopted in relation to the trunk. The infiltration of the homolateral vertebral plexus by 20 ml. of a 0.5% solution of procaine gave a very good result. The cochlear symptoms remained, and vertigo disappeared.

The acute influence of alcohol on hearing and the vestibular apparatus was investigated by Schwab and Ey.[255] Subjects with normal hearing and patients with conductive and perceptive deafness were selected. The patient was given 250 ml. of 35% alcohol, with a follow-up for 12 hours. Threshold and above-threshold audiometry was applied and repeated with every blood alcohol level determination. The vestibular apparatus was examined at the same time. The speech audiogram did not show any appreciable change up to a blood alcohol level of 1 per 1,000 above this level; differences showed up but never exceeded 10 db. The higher centers of listening, attention, and concentration were much more affected than the peripheral hearing organ. Regarding vestibular function, a direction-changing positional nystagmus was a nearly constant finding (90%). It becomes noticeable very early and as soon as the alcohol level decreases the direction of the nystagmus is reversed. Subjective experiences like vertigo, nausea, and vomiting are greatly accentuated under the influence of alcohol. Authors compare the acoustic and visual responses at different blood alcohol levels; hearing always proved more acute and prompt.

Alcohol intoxication and positional nystagmus due to it, is reported by Walter.[288] Every alcohol intoxication with a concentration of alcohol in blood serum of about 0.60% (drinking of 100-150 ml. 40% alcohol) gives a positional nystagmus which is observed under Frenzel's spectacles. It goes through three stages: (1) positional nystagmus; in the right side position nystagmus to the right and in the left to the left; duration about 216 plus or minus 50 minutes after drinking; (2) regulating time; no nystagmus; duration about 55 plus or minus 19 minutes; (3) positional nystagmus

II: nystagmus opposite to I and observed about 10-16 hours after drinking. This method of examination for alcohol intoxication is objective and of value in forensic situations. It gives, in addition to the examination of blood alcohol concentration, an objective picture of the patient's condition. The author discusses the errors of the method and points out the risk of examination during regulating time. He ends his paper with an account of nystagmus due to nicotine. This is always spontaneous and always of vertical direction.

Examination of seasickness by cupulometry is described by De Wit.[66] The slope of the curve is mainly determined by central factors. In normal persons the slope will become more horizontal on account of habituation, adaptation, or inhibition. In seasickness the slope remains steep. Further, specific seasickness with a steep cupulogram, unspecific seasickness, persons with deficient nutritional factors or suffering from Ménière's disease, are discussed.

The effects of cyclizine hydrochloride (Marezine) and chlorcyclizine hydrochloride (Perazil) upon vestibular function were studied by Gutner et al.[119] by means of the cold microcaloric and galvanic stimulation methods. Cyclizine in 100 mg. dosage notably decreased labyrinthine sensitivity. The results with cyclizine, 50 mg., and chlorcyclizine, 100 mg., were inconsistent and variable.

Caloric and rotatory labyrinthine tests, following the administration of various drugs used in the treatment of seasickness and given on the day prior to these tests, were studied by Garcia Vaquero Garrido and Ortega Garcia[101] in a number of young normal subjects. The action of the following drugs was studied: Vasano, Mareosan, Benadon (vitamin B_6), Marolin, Benadryl, and Phenergan. The first two are drugs containing atropine or its derivatives; the third is vitamin B_6, and the last three are antihistaminics. During the nystagmus, the caloric test showed less marked variations than the rotatory test. The results of the caloric test varied from 8.75% (Vasano) to plus 4% (Mareosan), and those of the rotatory test from −25% (Vasano) to plus 13% (Phenergan). In decreasing order, the degree of activity observed was as follows: Vasano, Marolin (dramamine), Mareosan, Benadryl, Phenergan, and Benadon. Drugs composed of atropine and, of the antihistaminics, Dramamine were the most active.

The treatment of motion sickness with antihistamine-amphetamine is reported by Arner and Diamant.[10] A new Swedish combined preparation containing 15 mg. Lergigan and 10 mg. amphetamine (Lergigan-Amfetamin, Recip, Stockholm) has been studied aboard a cruiser with a crew of 450 persons. Amphetamine used in this dose counteracted the hypnotic effect of Lergigan. The combination of Lergigan-amphetamine appeared to have all the qualities which would be demanded of a seasick remedy for naval use, not only because of its very great protection, but also because of its long duration of action and because of its slight side-effects.

Effectiveness of sodium bicarbonate injection was tested upon 152 persons who complained of chronic motion sickness. T. Muta, M. Muta, O. Muta, and Koizuka[204] suggest the effect of sodium bicarbonate might be attributed to the otolithic organ.

Control of nausea and vomiting and associated dizziness with Benadryl in 14 patients subjected to temporal bone surgery, 2 with labyrinthine hydrops, and 1 with suppurative labyrinthitis was tried by De Weese.[65] He concluded that Benadryl given intravenously is effective in controlling nausea and vomiting and subjective dizziness.

Toxic Labyrinthitis

Alajmo and Boccuzzi[1] have tested the action of streptomycin on chicken embryos. They injected several doses of the drug in different periods of incubation. Only very high dosages (50-100 mg.) produced damage to the organ of Corti of the newborn. Streptomycin proved to be more dangerous

than dihydrostreptomycin. There was no evidence of teratogenic action.

Dihydrostreptomycin is less injurious to the vestibular system than streptomycin but far more toxic to the cochlea, according to Minkenhof and Deinse,[196] who prove it statistically in 55 patients treated for tuberculous meningitis. They are convinced that all patients suffering from tuberculous meningitis should be treated with intramuscular and intrathecal injections of streptomycin. Repeated determinations of the streptomycin content of blood and C. S. F. (if possible once a week) are necessary in order to determine and correct a too large or too small dosage.

Robert [230] performed experimental studies on cats and also clinical examination in tuberculous patients treated with dihydrostreptomycin. The action of this drug upon the ear is marked by an initial stage of hypersensitivity, so that there is an abnormal increase of the number of nerve fibers excited by the sound. This explains the abnormal level of voltage of the action potential of the nerves. Early recruitment in patients can be explained by this increased potential detected in the animal. Then a real "acoustic trauma" appears with degeneration of the sensory epithelium and inner ear deafness followed by degeneration of the fibers and centers. Finally a great loss of hearing results from this progressive nerve atrophy.

A study is reported by Secondi [256] of the neurotoxic changes appearing in guinea pigs treated with large doses of streptomycin, administered at short intervals. Histopathological examination revealed marked changes in the cells in the region of the rhombencephalic nuclei of the acoustic nerve, while the cochlear and vestibular ganglia, the nuclei of the cerebellum and the hippocampus showed less marked changes. The important part played by certain factors in the localization and severity of the neurotoxic lesions is stressed. The most important of these are the concentration of the drug in the blood and the particular anatomical and physiological conditions prevailing in each region and depending upon changes in the permeability of the capillaries.

Ototoxicity from intermittent streptomycin therapy of pulmonary tuberculosis was studied by Cline, Houseworth, and Pitts.[52] Patients with pulmonary tuberculosis (100 cases) were treated six months with 2 gm. of streptomycin sulfate intramuscularly every third day and for two months with 1 gm. every third day. Serial audiograms and ice water vestibular tests revealed that 12% developed a slight degree of ototoxicity, 4% had moderate impairment, and 16% had marked dysfunction. The highest incidence of eighth nerve dysfunction occurred in the higher frequencies (4,096 and 8,192 cps) and in vestibular function. Marked toxicity appeared most frequently in the latter component. However, follow-up studies after eight months of streptomycin therapy revealed that most of the patients had only transient dysfunction regardless of the degree and type of ototoxicity observed during the period of drug administration.

To prevent the harmful effects of dihydrostreptomycin Masson [184] employs streptomycin sulfate combined with dihydrostreptomycin. The daily dose was 15 mg/kg. body weight. Of 122 patients so treated for three to six months, only four suffered permanent yet moderate loss of hearing.

Effects of dihydrostreptomycin on the cochlea are reported by Tionloc and Roxas.[274] Daily dosage varied from 0.5 to 1.0 gm. The least total dose was 40 mg. and the largest was 200 gm. The 14 cases studied showed a dip of more than 25 db. at 2,048 cps. In seven cases hypoexcitability by the caloric test developed without subjective manifestations of ototoxicity. The dip at 2,048 cps is a sign of warning of the onset of cochlear damage. Audiometric and vestibular examinations should be done before, during, and after therapy with the streptomycin group of drugs.

Dihydrostreptomycin is much more toxic to the auditory organ than streptomycin, states Aubertin.[16] In some cases there is

improvement in hearing after prolonged administration of vasodilator drugs of the vitamin B group.

Björk and Ranta [24] report a case treated with 1 gm. of dihydrostreptomycin daily for 10 days. Terramycin 250 mg. was then used q. i. d. for eight days three and a half months later. During terramycin therapy, a rapidly increasing tinnitus of the roaring type soon appeared. Hearing deteriorated for many months and appeared to be permanent. It thus appears possible that antibiotics other than those of the streptomycin group can cause perceptive deafness. It is advisable to discontinue these drugs in time if the patient complains of increasing tinnitus during treatment.

After reviewing some general information on nicotine damages, Maffei [178] describes his investigation of four groups of patients: 11 cases of amblyopia, 7 cases of cochleovestibular disturbances, 3 cases of vestibular lesions, and 3 patients referred to other hospital departments. In most cases the lesions affected the neuroepithelial structures of the ear, being accompanied by positive recruitment. The reversibility of milder lesions also suggested vascular damage. In cases of central disturbances toxic neuritis of the central pathways was found. A special test was used for the differentiation of reversible peripheral and irreversible central lesions: after abstinence the patient smoked two cigarettes. The audiograms taken just before and after the smoking showed typical increases of hearing loss in the cases of peripheral lesions.

Chenopodium oil is used as an anthelmintic. Toxic doses cause dizziness, vomiting, gastrointestinal disturbances, and loss of hearing. Since it is impossible to distinguish by histologic methods whether damage is limited to the organ of Corti or whether the ganglionic cells are also involved, Krejci and Bejdi [160] used radioactive phosphorus in a series of experiments with guinea pigs. Phosphorus is important in the metabolism of the ganglionic cells, and any change can be quickly spotted. In this manner it was possible to ascertain that chenopodium intoxication is limited to the organ of Corti, while the ganglionic cells remain unaffected.

Administration of quinine to man and to experimental animals (guinea pigs and cats) caused hypacusia and increase of Preyer's reflex threshold and of reflex contraction of the tensor muscle, according to Filippi.[85] These effects are not observed when an injection of atropine is given first; as atropine has the effect of inducing hypertension of the labyrinth, it is presumed that the effect of the quinine is based on a tension-increasing action of the labyrinth.

Toxic effects of quinine on the human labyrinth are known, but as regards the mechanism by which such effects are produced, opinions differ (spiral ganglion, Corti's organ, endolymph). Filippi and Finocchi [86] conclude from their experiments as described in previous papers that the most probable mechanism is that of pressure changes in the endolymph.

A review is given by Mazzei and Costa [190] of the known cases of occupational and experimental poisoning with petrol (gasoline), its mechanism of action and the routes of penetration and elimination of this toxic substance. Reference is made to a clinical case of chronic petrol intoxication with hypacusia of perceptive type, and a report is presented of the findings of experiments with rabbits poisoned for 90 days with petrol fumes, with a description of the histological lesions of the inner ear and of the bulbar nervous centres.

Vestibular Tests

Examination of the vestibular apparatus is incomplete if the test for positional nystagmus is omitted, states Cawthorne.[39] Two-thirds of patients with positional nystagmus do not exhibit any other signs of labyrinthine disease. When positional nystagmus appears any latent period between adopting the position and the onset of nystagmus, the direction, intensity and duration are noted. Two groups are recognized. In 10% it is of the central type: the nystagmus appears as soon as the head arrives in the

critical position and continues to beat at a steady rate as long as the head is held in that position, starts to beat again every time that position is resumed, often appears in more than one position of the head, and the direction of nystagmus may change with the direction of the head.* The other group is the peripheral (static labyrinth): latent period 2-10 seconds; eye movements tumultuous with wide excursions and combined rotatory and horizontal movements; dies down in 5-30 seconds (fatigable). In the peripheral group, the nystagmus is often only seen with the head in one position, almost always with the affected labyrinth undermost. Positional vertigo in the central group is rarely severe, whereas in the peripheral type it is much severer. In the peripheral group, some form of head injury seems to have been the commonest cause. Infection seems to be a factor also. In the peripheral group, in nearly one-third there were periods of remission during which the nystagmus could not be produced. In this group it is benign and often self-limiting.

Cawthorne [38] discusses positional nystagmus (p. n.). He examines the patients in a very simple way: to test for p. n. the patient is first of all seated on a couch. Holding the patient's head between his hands the examiner lays him back on the couch in the supine position with the head to one side. Afterwards the patient is brought upright again and after an interval of 10 seconds the same movements are made with the head to the other side and in a third experiment with the head straight back. P. n. is found to occur in two distinctly different ways. In one group, the nystagmus appears as soon as the head arrives in the critical position and continues to beat for as long as the head is held in that position. It will also start to beat again every time that position is resumed. Associated vertigo is often slight. The author found this to be the central type and saw it in only 10% of the cases of his series. In the other and much larger group there is a latent period of from 2-10 seconds. The nystagmus is often tumultuous but dies down in from 5-20 seconds. When the critical position is resumed anew, the p. n. is much slighter and finally disappears. This paroxysmal and fatigable nystagmus is the peripheral type. It has accounted for approximately 90% of the series, and of these not more than one-third yielded additional evidence of a labyrinthine lesion. The author prefers the distinction between the peripheral and central groups to the type distinction of Nylen, Lindsay, and others, since it is both simple and accurate and yields more information. P. n., especially of the peripheral type, is a disease of early to middle life. In many cases it is a self-limiting disease, and certainly in all the peripheral groups it is benign. In the discussion to this paper J. R. Lindsay stresses the importance of the finding that as many as 90% of the cases were of peripheral origin.

Aubry, Pialoux, and Bouchet [17] discuss the importance of positional nystagmus, apropos of the observations upon 128 patients showing this symptom. They divide positional nystagmus into four types: (1) spontaneous nystagmus of changing direction in different positions; (2) spontaneous nystagmus of changing intensity in different positions; (3) nystagmus appearing solely in certain specific positions; (4) nystagmus appearing in Rose's position. The examination does not avoid nuchal reflexes. The direction of the nystagmus was mostly horizontal but sometimes vertical or otherwise. Different subtentorial lesions often cause positional nystagmus together with other symptoms. Also, supratentorial lesions may provoke positional nystagmus but often as the only symptom and especially in Rose's position. The downward vertical nystagmus was most often found in these cases. The types 1, 2, and 3 are more often the sequel of subtentorial lesions.

The relation between the site of EEG abnormality and the presence of directional preponderance of caloric nystagmus was studied by Kirstein and Preber [159] in 68 patients with cerebral disorders. A directional preponderance was found in 29 cases, and

the EEG showed a focal abnormality in 25 of these cases. The focal abnormal waves were recorded from the frontal, temporal or parietoccipital regions, and lesions in these regions were in some cases subsequently verified at operation. In eight cases with normal caloric responses focal abnormal waves were recorded from either the frontal or the temporal region. It is concluded that directional preponderence in cases with cerebral lesions has no unequivocal localizing value, as has previously been claimed, and that the absence of directional preponderance does not exclude a lesion of either frontal or temporal region. It is pointed out that the presence of both directional preponderance to one side and a focal EEG abnormality on the same side indicates a cortical lesion in cases in which other signs of cortical damage are absent.

In a series of normal subjects, the caloric test by the method of Fitzgerald and Hallpike showed directional preponderance only in the form of small deviations from the equal response line according to Andersen, Jepsen, and Kristiansen.[5] A series of patients with intracranial lesions (without concurrent ear disease) were studied by the same technique. Directional preponderance was found to be very frequent in patients with supratentorial tumors, local vascular diseases, and head injuries, whereas it occurred with decreasing frequency in patients with encephalitis, multiple sclerosis, epilepsy, and migraine. No deviations from the control series were revealed in patients with cerebral arteriosclerosis, arterial hypertension, mental depression, or facial neuralgias. The delimitation between normal and pathological preponderance is discussed. Differences in duration of nystagmus to right and left of at least 30 seconds are regarded as pathological.

An attempt to clarify the clinical importance of the symptoms of directional preponderance is reported by Andersen.[4] Various intracranial disorders were studied. It was possible to demonstrate directional preponderance in 40% of cases with confirmed supratentorial lesions, but only if the lesion was unilateral or at least asymmetrical, and on such cases the preponderance was almost always directed toward the affected hemisphere. The pathology of the lesion did not play any role for the development of directional preponderance. The symptom was seen in patients with tumors, abscesses, thrombosis, and atrophic lesions. It may occur in undemonstrable conditions, such as vasomotor changes. Directional preponderance was seen in 26% of Ménière's disease and hence may be of labyrinthic origin.

Five cases of supratentorial affections of parietal location, whose clinical features pointed to a posterior fossa localization are reported by Marrero.[180] The syndrome observed in these cases were similar to the syndrome of frontal ataxia described by Bruns in frontal tumors. Modern investigation, however, goes to show that frontal ataxia is actually a disturbance of equilibrium due to affections of the supravestibular pathway. In the clinical picture there is predominance of disturbances of equilibrium, whereas there exists neither incoordination nor spontaneous oculovestibular disturbances.

Mittermaier and Christian[199] performed the caloric reaction test several times a day on a series of patients who never before had been subjected to vestibular stimulation. The routine technique consisted of injection by syringe of 10 cc. of 17 C water, in five seconds. At different times, the response varied greatly, not so much between individuals but rather between certain behavior groups. Fluctuations in the number of beats and amplitude were much larger than fluctuations in duration. The observation is very important that the stronger caloric stimulus increased frequency and amplitude more than the duration. For clinical purposes observation and report of the amplitude behavior must be considered of great significance.

Mittermaier[198] repeated caloric stimulation in the same ear at 15-minute intervals. He observed that repeated stimulation changes the outcome of the reaction. First

the amplitude of the nystagmus changes, subsequently its frequency. It is interesting that duration of nystagmus does not vary much. In a smaller group of patients the response was at first increased and later decreased; the larger group showed the opposite pattern of response. To report the medium average of amplitude and frequency has only limited value. The author feels that the quantitative and qualitative amplitudes should be registered by nystagmography, as they have real diagnostic significance, and this is the only way to study the slow component of the nystagmus.

Influence of the stimulus strength on frequency and amplitude in caloric nystagmus was studied by Christian.[49] Caloric nystagmus has been investigated by means of nystagmography in normal test subjects. The temperature of the water used in this test is of primary importance for the stimulus strength. The duration of irrigation and the amount of water play only a secondary part. The frequency and amplitude of nystagmus give more exact information about the strength of the caloric reaction than does the nystagmus duration.

An inverse caloric response (homolateral nystagmus) is occasionally elicited in normal subjects and more frequently in patients after otitis or head injuries. Riskaer and Permin[228] submitted 25 normal rabbits to caloric test by unilateral irrigation with 50 cc. of iced water with the following results: 11 animals (44%) showed heterolateral nystagmus alone; 12 (48%) transitory heterolateral nystagmus followed by intense homolateral nystagmus, and 2 (8%) first homolateral and then heterolateral nystagmus. Homolateral nystagmus was best elicited by irrigation with iced water. It was never elicited by water over 15 C. Repeated experiments with the same animal showed that the type of nystagmus was not constant, though the tendency to homolateral nystagmus seemed particularly pronounced in certain animals. Repeated irrigations more frequently gave homolateral nystagmus than did a single irrigation. Rabbits with full compensation after unilateral labyrinthectomy one or two months previously showed first heterolateral and afterwards intense homolateral nystagmus on irrigation of the intact ear with iced water. In these animals the diphasic reaction was constant. The results obtained highly suggest that the cold caloric test releases antagonistically acting nystagmus impulses in the labyrinth, though reaction due to cooling down of the flocculus cannot be left out of account.

An apparently normal caloric nystagmus is sometimes elicited from a destroyed labyrinth by irrigation with cold water. It is assumed that a latent nystagmus is activated by a sensitive reflex. Zwergius[307] concludes that the caloric reaction should also be done with hot water.

Temperature changes in the perilymph space in response to caloric stimulation in man were studied by Cawthorne and Cobb.[40] The subjects selected for the tests had unilateral Ménière's disease in whom labyrinthine destruction was to be done. Minute thermocouples activating a sensitive mirror galvanometer were used. On syringing the external auditory meatus for 40 seconds, with water either at 30 C or 40 C the following temperature changes were noted: (1) at the tympanic membrane the temperature changed to that of the water and after the flow had ceased it returned to its former level following a roughly expotential time curve (about 10 minutes); (2) the temperature inside the lateral canal showed little change during the first 10-15 seconds of water flow. The greatest rate of change usually took place between 20 and 70 seconds of the start of the flow, thereafter reaching a plateau in about 100 seconds. The peak value for water at 30 C was reached in 105 seconds, while at 44 C it was reached in 101 seconds.

Chilov[45] states that the intensity of vestibular reaction, especially of nystagmus, depends more upon the condition of the cerebral cortex than upon the peripheral vestibular organ. In rabbits, e. g., a caloric test under electronarcosis provokes no nystagmus, but as soon as the animal awakes it shows a nystagmus of the normal dura-

tion, which should have followed the caloric test in a non-narcotized animal. The biological sense of somatic vestibular reflexes can be understood, according to the author, only if the cortical reaction is primary. The somatic reflexes should be secondary. Nystagmus should be a conditioned reflex, because, in the period of evolution, animals rotating with their eyes open receive an unconditional stimulus of the retina, giving rise to a conditioned reflex when the eyes are closed. Taking away or doping the cerebral cortex in animals makes the duration of the caloric test much longer. In some patients a perrotatory nystagmus is of longer duration when they open their eyes, but in other patients the duration is shorter. This leads the author to the assumption of the existence of various groups of vertigo patients as regards somatic and vegetative vestibular reflexes.

It has been known for many years that repeated vestibular stimulations result in decreased response. Hood and Pfaltz [138] give a brief review of the work performed by others in this field. Rabbits were used as experimental animals. First the rotation stimulus was applied, and then the caloric stimulus. The allowed, calculated deflection of the cupula was 3 degrees with a 60 degree acceleration in one second. For rotation the Hallpike-Hood-Byford chair was used. The interval between experiments was from one minute to one week. Number of nystagmus beats, duration of nystagmus, and latent period before onset of response were reported and analyzed. The authors feel that there is some kind of cortical inhibition which may be called either fatigue, adaptation, or habituation, which causes the decrease of response. This assumption is mostly based on the fact that anaesthetized animals did not show the phenomenon of decreased response. In the investigation of caloric stimulation, 25 C water was used for 10 seconds. The vestibular response did not decrease as it did after repeated rotations. Caloric stimulation is unilateral and milder than rotation, and does not challenge central inhibition.

In a series of animal experiments Demetriades [60] demonstrated that caloric-rotatory-galvanic stimulation of the vestibular labyrinth causes a decrease of blood pressure. There is no fixed, predictable relation between the intensity of the stimulus and the drop of blood pressure. In Ménière attacks of hypertensive patients the drop may be considerable and may last several hours. The return to previous pressure is usually gradual.

The rotatory examination of the vestibular organ is described by Jongkees.[149] The possibilities and the dangers of the classical Bárány test are discussed. Cupulometry, the examination of the semicircular canal system with small regulable stimuli, is preferred on account of its greater usefulness, its lesser discomfort and danger for the patient, and its broader field of activity with smaller spread of the normal values. Based on sound mechanical calculation, cupulometry itself is a perfectly practical test, giving more information than the older one-stimulus tests. It may give information about rotatory sensation and nystagmus and it may differentiate between two types of dysymmetry (crossing and parallel lines) of the nystagmus. The method is still too new to have been tested exhaustively, but many interesting facts have already been observed in otosclerosis and head trauma, streptomycin intoxications, and fenestration cases.

The action of the various eye muscles during rotation is reported by Fermin and Jongkees.[82] By isotonic recording, the movement of the extraocular muscles in rabbits is examined during postrotatory nystagmus in various positions of the animal. The external and internal rectus muscles are more active than the other muscles in nearly every position (rotation about binaural-longitudinal-dorso-ventral axis). Perfect reciprocal action of agonist and antagonist is found in the external and internal rectus muscles when the rotation of the animal takes place about a dorsoventral axis; in other positions of the animal an alternating predominance of the function of one muscle

in a pair of antagonists is evident without reciprocity of agonists, i. e., contraction and antagonistic relaxation. The function of the internal and external rectus muscles is often thought to depend upon stimulation of the horizontal canals, and the function of other extraocular muscles should depend upon the activity of the vertical canals, but stimulation of the vertical canals alone provokes an action of the internal and external rectus muscles and stimulation of the horizontal canals alone makes the superior and inferior oblique and rectus muscles react as well.

Trincker, Bergmann, and Nehls [277] studied clinically the changes of postrotatoric nystagmus as a result of the changes of eye position. When a postrotatoric horizontal nystagmus was observed by fixing the eye in a horizontal plane and the eye was then directed 90 degrees vertically upward, the early nystagmus beating from one canthus to the other changed to rotatoric nystagmus. The reverse experiments gave corresponding results.

The optogyral illusion is of great importance in aviation medicine and for orientation in space. It is elicited by stimulation of the vestibular apparatus and is perceived by the tested person as an apparent movement of objects that are stationary. Van Dishoeck, Spoor, and Nijhoff [278] believe that it is a central process related to the turning sensation and occurring independently of nystagmus and kinesthesis. Their experiments prove that the illusion does not depend on a tracking of the image over the retina.

Investigations carried out by Aso [14] with nystagmographic recording showed the following: 1. Both amplitude and frequency of perrotatory nystagmus were greater in presence of optical stimuli (opened eyes in light room). Vision has a facilitating effect on the perrotatory nystagmus inasmuch as vestibular and optokinetic nystagmus superimpose each other. This is shown by the initial phase of nystagmogram, which is given by a rapid positive movement (as well as it occurs for optokinetic nystagmus) without any preceding slow negative phase. 2. Nystagmus produced by a long rotation (at the speed of one turn in 6 in.) decreases both in amplitude and frequency in absence of optical stimulations. When this latter interferes, however, the nystagmogram maintains uniform characteristics throughout, as it is observed in pure optokinetic nystagmus. 3. There were not essential modifications nystagmograms recorded during the influence of the two different types of optical interference (closed-eyes experiment in light room and open-eyes experiment in dark room) as well as between nystagmograms recorded in clockwise and anticlockwise rotation with unilateral optical stimulation. 4. Interference of optic stimuli during rotation produced increase in amplitude, frequency, and duration of postrotatory nystagmus. 5. When there is summation of optokinetic and perrotatory stimulation (the chair is placed in the center of the rotating cylinder, the inside of which is painted with black and white stripes, and both turn in the same direction) it has been found that when the speed of both the cylinder and the chair is the same, no nystagmus occurs, when the cylinder turns either faster or slower than the chair the nystagmus during the rotation appears in the reverse direction: the conclusion is drawn that the effect of optic stimulus is stronger than that of the vestibular stimulation. 6. Concentration of gaze leads to inhibition of perrotatory and postrotatory nystagmic reaction.

Eckel [70] placed microelectrodes in the medulla of rabbits to stimulate the vestibular nuclei, using for the first time bilateral registration. Short- and long-lasting rotatory stimuli were applied. Even during minimal stimulation, the effect could be registered. Different electrical responses in the nuclear areas of both sides were observed and followed. In some animals the nuclear areas reacted to acceleration on the same side, in others on the opposite side; further differences could be demonstrated in the first and in the second postrotatory phase. For instance, there may be an increase of activity in the first postrotatory phase fol-

lowed by a decrease in the second postrotatory phase, and vice versa. In stimulation of the horizontal canal, the utriculopetal deflection of the cupola was the activating, the utriculofugal deflection the inhibitory influence in the nuclear area. The position of the electrodes was marked by iron staining and histologic changes in the nuclear area could be observed.

Electronystagmography was used by Montandon [201] to register postrotatory and perrotatory nystagmus. The patient and the apparatus are placed on a rotating disc which may be operated at different constant accelerations or decelerations. Each rotation is divided into three periods: (1) progressive speed with constant acceleration, (2) constant speed, and (3) progressive braking with constant deceleration until stop. The threshold value at which nystagmus occurs for deceleration nystagmus is 1 degree/second^2. Higher values were found for perrotatory nystagmus (3-4 degrees/second^2). Normally the acceleration nystagmus lasted 90-100 seconds. No nystagmus was present during the second period. During deceleration nystagmus was opposite to, but of about the same duration and amplitude as that in the first period. Optimal nystagmus occurs at 3 degrees/second^2. Above this value (i. e., at even more marked acceleration) the duration of nystagmus is so long that it continues in the second period at acceleration and after total stop at deceleration.

Eye- and limb- or general nystagmus as affected by tactile or pressure stimulation on the skin was studied by Sato.[251] Electromyographically the tactile stimulation showed temporary marked augmentation of the discharge from the rapid contracting limb and eye muscles. The reverse and prolonged effect persisted during pressure stimulation. The above results showed that two components constituting the nystagmus behave quite differently from each other on skin stimulation, which obviously exerts a kinetic facilitatory effect on the rapid component and a tonic inhibitory effect on the slow component.

Equilibratory illusions in aviation is discussed by Pratt.[223] Vestibular stimulation in aircraft produces the same effects that similar stimulation would produce under any other situation. The illusory states produced by vestibular activity while flying are the result of capricious stimulation of the labyrinthine apparatus, with normal responses resulting from this sort of stimulus. The sensory responses with regard to aerial orientation are not only inadequate, but they are absolutely unreliable and must be completely distrusted if safety is to be maintained in the air. The only adequate devices for orientation in space are those mechanical devices regularly a part of aircraft equipment and designed to aid in flying blind. For proper equilibration two of the three systems (ocular, labyrinthine, and proprioceptive) must be intact and functioning.

In the air a pilot's spatial orientation is subject to illusory processes because (1) important visual clues normally used on the ground may be absent; (2) unusual accelerative forces may influence orientation; (3) there may be a marked conflict between the information derived from different sense organs. Clark and Nicholson [50] study the type of vertigo experiences encountered by flight students. A questionnaire asked firstly a description of the specific flight experiences and secondly the experiences of disorientation. In this analysis it was found that every vertigo item was noted by at least one student. In 62% there was an apparent turning when the instruments indicated straight and level. The investigation revealed that the commonest type of vertigo experienced in student pilots involved confusions with regard to altitude and position of the aircraft, although confusions with regard to visual perception were also common. The authors express the view that as vertigo is experienced sooner or later by pilots during training there should be a close relationship between it and aviation safety.

One hundred fighter pilots were examined otoneurologically by Aschan [13] in order to determine whether their profession

influenced their otoneurological status. Fully trained fighter pilots with daily ascents show a sensation cupulogram with a higher threshold value and slighter inclination of the curve of the sensation cupulogram than do pilots with less training. The nystagmus cupulograms show the same. In those there is often directional preponderance and the pilots concerned more easily rotate their aircraft in agreement with this directional preponderance. Absence from flying for a period of months leads to a normalization. Changes in calorigrams could not be demonstrated. The genesis of this vestibular fatigue or adaptation demonstrated in "normal" fighter pilots must probably be sought, not peripherally in the cupula itself, but in the nervous part of the vestibular apparatus.

Krijger [162] has recorded the linear accelerations of the aeroplane during aerobatics. The impulses sustained by the semicircular canals and otolithic organs during these performances are analyzed. Their relation to the occurrence of spatial disorientation and neurovegetative disturbances is discussed. The author postulates that a normally functioning labyrinth is desirable for proficiency in flying. To examine this function, the Bárány test is rejected as being unphysiological and unrevealing. The cupulometric method of exploring the equilibrial functions is the method of choice. Moreover, it holds promise as a preselective test in singling out those candidate pilots who are liable to fail for reasons of unsurmountable air sickness.

Browne and Beck [33] report an investigation of 50 consecutive men possessing certificates of disablement from coal-miners' nystagmus and 47 randomly chosen miners without the disease as controls. The mean ages of the two groups were 52 years 8 months for the group with nystagmus and 51 years 2 months for the controls. Previous illnesses were almost identical for the two groups. Pulse rate, respiration rate, and blood pressure tended to be higher in cases of nystagmus. The onset of nystagmus was associated with rapid promotion to face work or to more responsible positions. The age-group, 15-24, had spent 3.3 years on face work; the controls 2.2, and a similar trend was shown in the next two decades. In the decade 35-44, the men with nystagmus spent 0.7 years as deputies; controls 0.5, and this ratio roughly held for the next two decades.

A case of fistula in both vertical canals as well as a thin fistula in the promontory in which vertical nystagmus was released indirectly was reported by Lund.[177] The fistula symptom released from a fistula in a vertical canal is horizontal rotatory. Among 22 cases with a fistula sign released from a fistula in the horizontal canal there were 5 cases with contralateral nystagmus indicating that the function of the horizontal canal had been destroyed. Varying direction of a fistula sign during a test indicates a fistula into the vestibule. The fistula sign is a general labyrinthine response elicited from the maculae and cupulae by altered pressure inside the labyrinth.

Latent nystagmus and alternating hyperphoria is reported by Anderson.[6] Of 34,000 patients, 138 were found with congenital nystagmus, of whom 20 showed "latent nystagmus." This is an uncommon and unexplained condition, wherein nystagmus, not normally present with both eyes open, becomes manifest on occlusion of one eye and, as a rule, on conjugate deviation. The nystagmus is usually rhythmic in nature. As with experimental vestibular nystagmus, the exciting agent appears to be asymmetrical stimulation of distance receptors, but the former condition is physiological whereas latent nystagmus arises from a disordered basic mechanism. With one exception, every case of latent nystagmus in this series showed alternating hyperphoria. Both conditions can be increased by nervous tension and fatigue. The nystagmus on occlusion is nearly always rhythmic, with the initial slow component toward the covered eye. The nystagmus that occurs on conjugate deviation is frequently rotatory; when it is rhythmic the slow component is away from the direction of gaze. It is suggested that latent nystagmus and alternating

hyperphoria may be due to arrested development of the oculomotor mechanisms that underlie binocular fixation. Their full elaboration may be prevented at any early stage in development when they are vulnerable. It is suggested that difficulties at birth, which were very common in this series of patients, may cause both these conditions. No evidence of inheritance was observed in this series. All the patients in this series and the great majority in the literature were under 35 years of age. Latent nystagmus appears to grow less with age in some patients, but no diminution of alternating hyperphoria was observed.

Kataoka [153] performed Ashibumi-Kensa for 113 deaf children, making them stamp their feet 100 times. The results are as follows: 1. Among deaf children are a few who got good results from Ashibumi-Kensa; there are many, however, who showed poor results. With regard to those who showed poor third degree, totally deaf children are more numerous than children with residual hearing. 2. Among totally deaf children, there are a few who show deviation, instead of their physiperceptible physical shaking. This is why their bilateral vestibular function is severely damaged in equal degree. It can be easily explained by the outcome of rotation test; both the rate of bilateral non-nystagmus and ears non-responsive being in high figures. 3. Results of Ashibumi-Kensa are in some degree proportional to those of goniometry; however, in a certain case, it is not so. In approaching the state of vestibular muscle tonus of lower extremities of deaf children, Ashibumi-Kensa should also be carried out. 4. It is recognized that there is a certain relationship between it and acoustic function. 5. Those with poor results of Ashibumi-Kensa were found more numerous among acquired deaf-mutes than among congenital deaf-mutes.

Toshihiro [276] provoked nystagmus in guinea pigs by irrigating the external ear canal with chloroform, and, having killed them by decapitation during the first, maximum, and terminal period of nystagmus, observed the histological changes of the macula and crista. The nystagmus produced when the ear canal is irrigated with chloroform is toward the opposite side. It is difficult to find a clear picture of labyrinth hydrops during nystagmus. The cupula of the irrigated side is smaller than that of the opposite side, or, rather, the latter swells. The nuclei of the sensory cells change with nystagmus; on the whole, the round nuclei gradually increase from the first period till the maximum, and diminish toward the terminal. The macula does not present the same findings as the crista.

The perrotatory and postrotatory nystagmus in pigeons was studied by Van Eyck [279] following lateral labyrinthectomy by means of electromyography of the cervical muscles (by insertion of two pairs of electrode needles into the exposed muscles on both sides). The animal was placed in a Gerebtzoff apparatus, with the head fixed into such a position that only the horizontal canals were stimulated. Rotation was effected by hand (five times within 10 seconds), and abruptly arrested. Sixteen animals were examined. Anticlockwise rotation of normal animals was followed by the occurrence of rhythmic perrotatory contractions in the right muscle mass and upon arrest (postrotatory) in the left. No perrotatory muscular contraction (ampullofugal stimulus on the right) occurred 20 minutes after destruction of the left labyrinth. Sudden arrest (ampullopetal stimulus) did give rise to muscular contraction. Perrotatory stimulation 24 hours after labyrinthectomy caused a weak irregular muscular contraction; five months later the reaction was again as marked as prior to operation. No hypothesis can as yet be made which affords an explanation of the mechanism of compensation.

Anatomy and Physiology of the Ear

An extensive study has been made by Porta [219] of the auditory apparatus, from those of the lower classes of animals to the human ear, and the anatomical and physiological data concerning the structure

of the various auditory apparatus are analyzed. Some primitive form of an auditory-static organ, involved in both static and movement functions, is already found in Phytozoa, molluscs, and gastropods. In some of the Crustacea auditory sacs were found, containing sensitive hairs which can be stimulated by sound. In insects, there are cordotonal organs and tympanic organs composed of nerve fibers; they lie in the antennae and in the wings, and make an auditory function possible; sometimes these organs are simultaneously adapted to hearing and to palpation. In the vertebrates, the auditory organ has already been subdivided in different parts with corresponding functions. In fishes, for example, we find an internal ear consisting of a superior and an inferior labyrinth, with respectively a static and an acoustic function, and a middle ear which consists of the swimming bladder. The external ear is not yet represented. Frisch and Stetter have studied fishes by the method of conditioned reflexes and have demonstrated that fishes have not only acoustic sensitivity but can also to a certain degree analyze tones. In Amphibia the hearing organ is better developed than in fishes; there is already a beginning of the external ear, viz., a small lateral fossa closed by a tympanic membrane. A real external ear is found in birds; the middle ear is very complicated, and the inner ear is comparable to that in mammals. Birds have a very well-developed hearing capacity and are capable of perceiving the slightest variations in frequency. In mammals, the auditory organ has reached a maximum perception; all parts are well developed and have acquired their final form. A review is given of the very interesting studies of Weber, Engelman, Galambos, and Davis, which have been predominantly concerned with study of the hearing capacity in dogs, cats, and other mammals. From these studies, it was concluded that many animals (dogs, bats, and some of the birds) have a much more perfect auditory function than man, perhaps in connection with special requirements deriving from their way of life, or to offset the scanty development of other sense organs (bat). The author concludes this brilliant phylogenetic study by expressing the wish that many more detailed studies will be made of the auditory organ in mammals, both to throw light on problems of malformation, and to improve on our present functional surgery of the ear.

The oval window has a more complicated structure than is usually assumed, because there are several adjustments to reinforce the stability of the stapediovestibular junction. Brunner[34] states that these adjustments act to keep the force exerted by the stapedius muscle in physiological limits. In other words, they are antagonists to, and also moderators of, the function of the muscle. The following structures serve this purpose: (1) the cartilage at the frame of the window and at the footplate of the stapes; (2) the spiral rotation of the anterior part of the window frame; (3) the auxiliary reinforcement ligaments; (4) the connective tissue of the fistula ante fenestrum (perhaps not constantly). The stapes is very well fixed in the oval window. For this reason it must be difficult to remove the footplate by pulling on the crura. Incision of the anterior portion of the annular ligament would facilitate its removal.

Anatomic study of the lamina spiralis in its vestibular portion is reported by Mounier-Kuhn et al.[202] The hook-like gap formed by the osseous spiral lamina in the dry bone appeared to be closed by a firm membrane in the fresh specimen. This membrane, which is continuous with the basilar membrane, prevents the labyrinthine fluid from passing from one scala to the other in the vestibular part of the cochlea, thus leaving the helicotrema as the only route of communication between the two scalae.

A light and electromicroscopic study of the structure and innervation of the cochlear hair cells is reported by Engström and Sjöstrand.[74] The organ of Corti is innervated by both afferent and efferent nerve fibers. The ganglion cells of the afferent fibers are situated in the spiral ganglion

while those of the efferent fibers emerge from the contralateral superior olive. The nerve fibers lose their myelin sheath in the osseous spiral lamina on their way to the organ of Corti. They then continue their course, unmyelinated, through the medial attachment of the basilar membrane to the inner and outer hair cells. The distal endings of the nerve branches are club-shaped and settle close to the plasma membrane of the inner and outer hair cells; they are separate from one another. They vary in size and number but are similar in general structure. They are enclosed in a thin plasma membrane. The nerve endings seem to be largest under the cells in the first row of hair cells and smaller under the outer hair cell rows.

Using Weber's histological technique, Portmann and Portmann [221] find the organ of Corti innervated by a double system of afferent sensory and efferent fibers, the latter originating from the medulla and ending at various turns of the cochlea leveling with the internal spiral plexus. Formal proof of the efferent nature of these fibers is given morphologically, although their function has not yet been established. Theoretically they are considered, according to Fernandez, to constitute the anatomical substratum of cochlear interactions.

A survey is given by Barcia Goyanes [18] on more recent modifications of the knowledge regarding the auditory pathways, with reports on many details. The trapezoid body in its quality of an authentic auditory chiasma has gained more importance in the light of new observations. Attention is given to the connection of the auditory pathways with the cerebellum. There is in the cerebellar cortex a double representation of the various parts of the body in the form of mirror images. Well known by observations in animals, these conditions are only assumed in man. It would be of outstanding clinical importance to clarify the coordination of the exteroreceptors with the cerebellum. Animal experimentation discovered the existence of a secondary auditive zone, and in man a secondary auditive area was demonstrated. Tonotopical experience, although gained solely on animals, gives a good basis for the assumption that in man, too, a general law or organization of the auditive cortex is obeyed.

With the aid of a strict experimental technique, an attempt was made by Botner and Pivotti [29] to determine whether, across the middle wall of the tympanic cavity, anastomoses exist between the arterial vessels of the middle ear, which are branches of the carotid artery, and those of the internal ear, which are branches of the vertebral artery. The experiments were carried out in dogs and guinea pigs, and the findings served to show that no such anastomoses exist. It is improbable that there is a functional correlation between the vasomotility of the middle ear and that of the membranous labyrinth.

Important observations on the circulation in the cochlea made from exposure of the spiral ligament in living animals are reported by Seymour.[259] Prolonged sympathetic stimulation produced marked concavity of Reissner's membrane in 7 of a group of 19 animals. This is found only on the side of stimulation and it is suggested that the dipping is due to a decreased secretion of endolymph secondary to a decreased blood supply. Flow of blood through the stria vascularis is influenced by (1) the state of the precapillary sphincter, (2) by the angle at which the capillary arises from the metarteriole, and (3) by the damming effect of specialized draining vessels in the basal turn of the cochlea which are described for the first time. The slow stream in the stria gives time for the maximum oxygen and metabolic exchange to occur between the blood and endolymph, a condition essential for the well-being of the organ of Corti, since this structure is not provided with vessels of its own. Since the strial network is continuous from apex to base, with few vascular connections between these two points, it follows the oxygen tension will tend to fall from apex to base and that blood-borne toxins will tend to concentrate at the base. As to the saccus endolymphati-

cus, its function is to secrete endolymph. Endolymph is formed in three and probably four ways. Two occur in the region of the intermediate part of the sac from two types of papilla found there, one with a vascular and the other with a connective-tissue core. The surface epithelial cells distend and rupture discharging their contents into the endolymph. A third way occurs in the epithelium at either end of the rugose portion, where the surface cells protrude a clear globule which is liberated from the cell. Finally, the distal part of the sac secretes a watery solution of low electrolyte content. The high concentration of potassium in the endolymph (20-30 times higher than that of a filtrate such as perilymph) indicates that it is a secretion formed largely of cytoplasm from living cells. The saccus is not the only source of endolymph, the fluid being secreted by the stria vascularis, the maculae of the saccule and utricle, and from the cristae of the semicircular canals. The production, circulation, and absorption of endolymph are largely confined to each compartment of the endolymphatic system. Perilymph is formed largely from the blood as an ultrafiltrate from the cerebrospinal fluid and from endolymph. Its absorption is into the veins, similar to the absorption of tissue fluid. Because of its small size, the internal auditory artery is predisposed to all the vascular hazards which contribute to perceptive deafness. In many instances Ménière's disease may have an underlying vascular cause. Intralabyrinthine pressure may be reduced initially because of reduced blood supply. This reduces the secretion and alters the composition of the endolymph and causes an accumulation of metabolites. The hydrops then follows in the later stages.

Direct microscopic observations of the blood vessels of the spiral ligament are reported by Weille et al.[290,291] Arterioles, capillaries, arteriovenous arcades, and venules of the spiral ligament, and capillaries of the stria vascularis were observed. The arterioles, arteriovenous arcades, and venules possessed the ability to contract and dilate independently. Intermittence of linear blood flow was observed in arterioles, arteriovenous arcades, capillaries, and venules. Rate of blood flow varied from time to time in each type of vessel.

According to Weille et al.,[292] anesthetized guinea pigs sensitive to egg white can die in two to three minutes after the shocking dose of antigen. The changes noted in the blood vessels of the spiral ligament included constriction of arterioles and initial constriction followed by dilatation of venules. When death did not occur until several minutes after the shocking dose, emboli and thrombi were seen in both arterioles and venules. When the guinea pig recovered from anaphylaxis, thrombi in the vessels did not disappear.

Examinations of patients with partial or total uni- or bilateral and combined destruction of the vestibular and cochlear function caused by cerebral concussion, cranial fracture, treatment with streptomycin, or a congenital condition led to the following conclusions by Braylovski[31]: Nonexcitable vestibular apparatus does not affect the threshold of sound intensity necessary for evoking the cochlear-pupillary reflex. The reflex is produced by sound stimulation corresponding to the individual hearing threshold; hence the term cochlear-pupillary reflex is justified. In cases with destroyed cochlear function and unaffected vestibule, the reflex may be produced by very intensive sound stimulation.

The vibration of the foot of the stapes in man was studied by Parow.[215] Attempts were made to determine the position of the axis of rotation of the foot of the stapes and so on the perilymph displacements in the internal ear; the extent to which the tilting movements described by Békésy also occurred in this test arrangement was studied as well. The material consisted of 25 freshly obtained human petrosal bones, in which the elasticity is maintained relatively long (until the onset of tissue decomposition). Frank's method of recording the movements of the plate was used; this method involves photography of the so-called Lissajous figures, which occur when a pin-

point light beam is directed across two mirrors with perpendicular axes of rotation. One mirror is fixed on the membrane of a 10 watt permanent magneto loudspeaker (registration of tone in the horizontal axis), while the other one is mounted on a three-legged stool, one leg of which is placed on the plate at various points, whereas the other two rest on the petrosal bone; this mirror diverts the beam into the vertical plane. The investigations showed that, within a single movement to and fro, the plate carries out complex composite movements. In addition to a translatory movement, rotatory movements occur on three axes perpendicularly placed, i. e., two horizontally perpendicular to each other and a third perpendicular to the two former. The latter showed a free center of rotation, which, at low intensity of sound, is closer to the fenestra ovalis than at high intensities. The perilymph displacement can be calculated. The Békésy movements were seen but could not be interpreted with certainty. The position of the foot of the stapes in space can be determined exactly, at each point of time, by means of the maximal amplitudes (dyne/sq. cm.) and the mutual phase difference of the separate points palpated.

The right stapedius muscle was sectioned in the middle ear of 24 rabbits. Subsequently 100-400 rounds of explosions of 100-130 phons were set off 15 cm. in front of the animals at two-second intervals. Histological examination, according to Taruya,[270] demonstrated that the damage to the organ of Corti was least in the basal turn and greatest in the second turn. Likewise, it was clear that the side with the sectioned stapedius muscle showed more damage than the other side. It was also clear that the degree of damage following section of the stapedius muscle is less than that following section of the tensor tympani muscle.

Studies on the function of the tensor tympani muscle were made by Okamoto, Sato, and Kirikae.[210] The latency in this reflex was measured as follows: burst of action current of the muscle 6-8 msec., contraction of the muscle 7-10 msec., and inward movement of the tympanic membrane 18-19 msec. From the behavior of the discharge, the components of this muscle may be divided into two neuromuscular units, tonic and kinetic. The threshold of activity of the tonic unit was 80 phons and that of the kinetic unit was 90 phons. The fluctuation of the tympanic membrane in the retracted position under continuous loud sound is due to the synchronous discharge of almost all the neuromuscular units of the tensor tympani muscle. Acoustic stimulation of one ear evokes reflex contraction of the tensor tympani muscles of both ears. The significance of the tensor tympani reflex is not clear, but supposedly it protects the inner ear against acoustic injury and accommodates it to acoustic stimulation.

The destructive changes induced in the cochlea of rabbits by pure tones of 3,500 cps, 100 phons, and 250 cps, 115 phons, respectively, were observed by Tomoyuki.[275] The right m. tensor tympani was dissected, the left one maintained intact as control. At both frequencies, the cochlea of the side operated on is more severely injured. This is considered as evidence for the protective function of the tensor tympani muscle. The operation has no influence on the localization of destruction in the cochlea.

Some electromechanical properties of Corti's organ are reviewed by Békésy.[21] The drumhead with the ossicles is a better transmitter than the usual telephone receiver. Cochlear oscillations were studied by direct microscopic observation and registration of microphone potentials. A cochlear model demonstrated that the cochlea is a complex structure of isolating membranes with intermediate layers of conducting fluids. Microscopic observation revealed wandering waves which are progressively shortened toward the helicotrema. From a certain minimal length on they are functionally insignificant. The direction of oscillation of the cells of Corti's organ changes. They first oscillate perpendicularly from the stapes onto the helicotrema, then parallel with the cochlear axis, and

finally in the direction of the length of the basilar membrane. Since the auditory organ is highly sensitive and since the sensory cells as firm bodies require a certain force to be stimulated, a transformer must be available which transforms the slight fluid pressures into more marked forces applied to the cell surfaces. It is assumed that shearing forces are involved. The mechanical energy of the oscillation is not transformed into electrical energy of the microphone potentials since disappearance of microphone potentials (in case of oxygen deficiency) has no effect on the oscillations. These produce the microphone potentials by a trigger mechanism; the energy of these potentials is derived from direct current tensions present in the organ of Corti.

The excitation of nerve impulses in the cochlea is discussed by Davis.[56] Nerve impulses are probably excited by the cochlear microphonics. The action potential of separate nerve fibers of the acoustic nerve was obtained by Tasaki. For each fiber a frequency was found which had the lowest threshold of excitation; it was found, however, that below this frequency the threshold was only slightly increased, while a marked increase was seen above it. Similar results were obtained from the cochlear nucleus. The endolymph of the scala media is markedly positive, electrically, as compared with the perilymph. By replacing perilymph by a potassium-rich Ringer solution, the electrical responses in Corti's organ are considerably altered. This leads to the conclusion that the tunnel and Nuel's space must be filled with perilymph instead of endolymph. The ion-permeable layer is therefore not the basilar membrane but the lamina reticularis. The nutrition of the organ of Corti, including the hair cells, is derived from the scala tympani and not from the scala media. If the tunnel of Corti were filled with fluid of the chemical composition of endolymph, the nerve fibers which are immersed in it would not be able to conduct nerve impulses.

After a survey of the modern theories of hearing, a personal theory is described by Borghesan,[28] which is primarily based on extensive anatomical and histological examinations. The cilia of the sensory cells continue with a long, elastic portion into the tectorial membrane, so that sensory cells and tectorial membrane form an anatomical and functional unit. The cilia of the external auditory cells are localized at the vestibular surface of the tectorial membrane; those of the internal auditory cells at the tympanic surface. The mechanical oscillations first affect the vestibular surface, which is very easily deformed, and involve the deeper layer of the tectorial membrane only in greater intensities. Accordingly, the external auditory cells are first stimulated, with increasing intensity the other ranges of external hair cells, and finally the internal hair cells. Thus, the intensity of the oscillation is represented by the number of sensory cells involved, but the frequency by the position of the stimulated sensory cells in Corti's organ. In this respect, so-called "syntonic sectors" can be distinguished, i. e., groups of sensory cells responding to a certain frequency, in which the intensity determines which cells of this group are stimulated and how many. At threshold intensity, only the cell at the top of the syntonic sector responds, which is formed by one of the outermost auditory cells. With increasing intensity, the other sensory cells are also involved, finally the internal auditory cells, which form the basis of the sector. With the presented considerations, most of the physiological and pathological phenomena of the hearing process can be explained.

Regional hearing losses from induced cochlear injuries were studied by Sutton and Schuknecht [269] in experimental animals. Cortical hearing tests were used before and after the cochlear injury. A quantitative study was then made of the inner ear pathology. The results confirm previous concepts of an orderly, spatial distribution of frequencies within the cochlea. The sharpness of tuning is greater for high

frequencies than low frequencies. Success in creating more restricted threshold losses was interpreted to mean that a greater burden for frequency analysis must be placed on a place principle of cochlear function than has been demonstrated previously.

Operations on the vegetative nervous system have been performed in recent years to treat some aural disorders. This treatment is based on the supposition that by interruption of vasoconstrictor fibers, the blood supply on the inner ear can be improved. Krejci and Bornschein [161] in experiments on 11 cats found no evidence for the supposition that the cochlear microphonic potentials are influenced by prolonged sympathetic stimulation.

An attempt has been made by Goldstein [110] to study further the properties of the summating potential (SP) and to clarify its relationship to the action potential (AP) and the cochlear microphonic (CM). The electrodes were silver wire infixed in scala vestibuli and scala tympani of the basal turn. The sound-generating and recording systems were the same that Davis has used. "Tone-pips" of the frequencies 500, 2,000, and 8,000 cps were pulsed at 20 per second. In scala vestibuli, the 8,000 cps pip produces an SP that is negative relative to the neck, and the 500 cps pip produces a positive one. The corresponding SP recorded from scala tympani each have the opposite sign. There is no apparent SP in either scala in response to the 2,000 cps pip. With death from asphyxia, the SP of the scala vestibuli becomes positive for all frequencies and then later becomes negative for all pips. The electrical polarities are always opposite in scala tympani. Masking experiments were made by presenting a white noise simultaneously with an 8,000 cps pip. The AP was greatly reduced; SP was also reduced but not so much; the CM was not affected. After trauma of the cochlea (acoustical, chemical, and surgical) SP in scala vestibuli becomes negative for all frequencies. The author suggests three components of the SP with varying resistance to lack of oxygen and damage. One of these components is the nerve action potential of the nerve fibers within the organ of Corti. The origin of the others is as yet unknown (the author's original suggestion was that the external and internal hair cells were the sources of all components).

Some original hypotheses regarding the nature of the so-called cochlear electrophonic effect are suggested by Chocholle.[48] The electrophonic is not believed to be due to fundamental electrical phenomena, as the ear does not contain any structures capable of transforming electrical energy into sonorous energy. In addition, in view of the distance between the tympanic membrane and the windows of the inner ear, the electrophonic effect cannot be attributed to electrostatic phenomena. Finally, it is stated that the electric current cannot reach the inner ear. Accordingly the electrophonic effect obtained in normal conditions should be differentiated from that obtained in pathological conditions. When an electrode is inserted into the middle ear, the electrophonic effect appears at the level of the mucosa lining the middle ear; when the electrode is placed on a window, however, the effect arises at this window or in the structures of the inner ear.

Electrical responses to acoustic clicks from various way stations of the cat's classical afferent pathway are described by Rosenblith.[238] The concept of probability of response is introduced as a key element in the interpretation of the electrical activity of populations of neurons. Intensity functions are presented in which both the amplitude and the latency of the first neural response and of the cortical response are plotted versus click intensity. Neural responses to pure tones are briefly discussed. Experiments are summarized in which the behavior of the first neural response to clicks is studied (*a*) against a background of white noise ("masking") and (*b*) after exposure to intense sounds; also experiments in which responses to pairs of clicks were recorded simultaneously from the

cat's round window and auditory cortex. Finally there is a discussion of the possible relations between data from auditory electrophysiology and those responses of the total organism that are commonly associated with the word "hearing."

The cochlear potentials produced in cats by pure-tone stimuli of 250 to 8,000 cps were recorded by Goto and Hibino.[117] Tests were made with the tympanic membrane intact, after perforation of the membrane with a hot needle in the anterior, posterior, or umbo region, and after progressively enlarging the perforations until the membrane was entirely destroyed. Although losses for every frequency tested occurred regardless of the location of the lesion, it is concluded that the posterior region of the tympanic membrane is more important than the others for frequencies below 3,000 cps, while at 8,000 cps (and probably higher) the anterior region of the membrane is more important than the other regions. Very small lesions in any region were sometimes found to cause a small loss in response, sometimes no loss, and sometimes even an improvement in response.

Experimental and clinical studies on the change in content of some enzymes in the labyrinthine and the cerebrospinal fluids, as effected by the injury of the auditory organ are reported by Ito.[144] Amylase content of the CSF in the perceptively deaf was invariably lower than that of the normal, but catalase was absent in the CSF of normals and the perceptively deaf. In normal and experimentally damaged animals amylase content of the labyrinthine fluid was always found higher than that of the CSF. Amylase in both cerebrospinal and labyrinthine fluids was definitely reduced in cases of experimentally damaged auditory organs. Catalase was virtually absent in the labyrinthine fluid or CSF of normal rabbits, but was frequently recognized in both fluids of the experimentally damaged auditory organ.

Ninety patients were exposed by Ferrari[83] to hyperoxygenation and tested audiometrically. Normal persons showed no change in their audiograms. Hearing losses of vascular, toxic, or presbyacustic origin remained unimproved. There was no influence on auditory fatigue. However, there appeared to be a difference between old and recent cases of cranial trauma. While cerebral damage was still in the stage of improvement, oxygen exposure improved the loss for high tones.

The effect of oxygen lack and decreased blood pressure on the microphonic response of the cochlea was studied in guinea pigs by Gisselsson.[108] Cochlear potentials are intimately related to the activity of the hair cells (demonstrated only in the presence of intact hair cells). The amplitude of cochlear potentials is decreased by oxygen lack as well as by extreme fall in blood pressure. Oxygen lack produced by potassium cyanide (which inhibits cellular respiration but increases blood pressure) decreases the amplitude of cochlear potentials. Oxygen restriction produces an initial decrease in cochlear potentials and a simultaneous transient slight drop in blood pressure. After one to two minutes, the blood pressure increases with a further decrease in cochlear potentials. Subsequent supply of oxygen increases the blood pressure violently and the cochlear potentials too. The blood pressure then gradually drops with a gradual decrease in cochlear potentials. This biphasic decrease in the amplitude of cochlear potentials suggests that the potentials are made up of two factors, one sensitive to oxygen lack, the other to changes in blood pressure. The cochlear potentials are extremely sensitive to oxygen lack, about 30 seconds' deprivation of air being sufficient to produce reversible injury to the cochlea, 2 minutes' deprivation being enough to cause irreparable damage to the organ. Cochlear potentials are the expression of a biological effect and not of a piezoelectric or other effect.

The correlations between behavioral audiograms and recordings of cortical potentials in experimentally deafened cats was studied by Hind and Schuknecht.[132] Cortical

thresholds were defined as the minimum intensity of tone impulses capable of eliciting strychnine potentials. Curves expressing threshold elevation with respect to normal animals were prepared for both the behavioral and the cortical responses; these displayed above charts of cochlear histopathology. A mean value of 3.0 db. greater loss for the behavioral test was obtained when the differences between corresponding pairs of behavioral and cortical threshold elevations at all frequencies in all animals were average algebraically. The standard deviation of this collection of threshold differences was 17.4 db. Hypotheses advanced to explain the discrepancies include the effects of: (1) uncontrolled physiological variance in the cortical recording procedure; (2) the fact that the absolute cortical threshold exceeds the absolute behavioral threshold by 30 to 40 db.; (3) the use of an abrupt onset for the cortical tone pulse. It is concluded that the cortical test is the most practical and descriptive electrophysiological indicator of threshold cochlear function at present available, although it cannot be considered a precise substitute for behavioral threshold determinations.

Nakajima[205] intended to clarify the mechanism of the auditory disturbance from the viewpoint of humoral pathology. Following up changes of pH of the cerebrospinal and the labyrinthine fluids in perceptive deafness as well as studying experimental deafness in animals, he established a definite difference in pH existing between pathological and normal conditions. The pH of both fluids had a tendency to become normal when various therapeutic treatments were applied through the CSF. The author lays emphasis on his data in discussing disorder of the auditory sense, and suggests that attention should be directed to the properties of the labyrinthine fluid also in conductive deafness.

The distribution of small vessels in the spiral ligament and spiral lamina of the cochleas of the guinea pig, the cat, and man is very similar. The arterioles radiate out from the modiolus over the scala vestibuli and ramify in the spiral ligament, reports Smith.[265] Some of the branches turn in a spiral direction above Reissner's membrane and form a network in the upper spiral ligament. The stria vascularis is a separate capillary network with arterioles entering and venules leaving it at intervals. A single vessel is found in the spiral prominence. In the spiral lamina, the vessels in the limbus form a subepithelial spiral border at its edge. The radiating nerve fibers are well vascularized. The capillary field ends laterally as a plexus in the labium tympanicum which may extend out to the tunnel of the organ of Corti. The spiral ligament has several distinct capillary groups. The networks in the stria vascularis, the spiral prominence, and the upper and lower spiral ligament are separated by vascular supply and drainage. Such a pattern greatly favors the possibility of regional circulatory variations. Not only is it probable that there are differences in pressure and velocity of flow but circulatory disturbances may be found in one place without necessarily being present elsewhere. It seems possible that perilymph is formed and resorbed in the cochlea by the capillaries in the spiral ligament and spiral lamina.

In animal experimentation and in patients Hasegawa observed disappearance of the otolith crystals after intravenous administration of sodium bicarbonate. To gain more information investigations were carried out by Hiroshi and Juichi[134] without decalcification of the labyrinth on guinea pigs and rabbits. It was found that sodium bicarbonate and sodium citrate did not dissolve the otolithic crystals after intravenous injection of even high doses. In further experiments otoliths from man, rabbit, guinea pig, rat, frog, and mackerel were submitted to Meigen's reaction, electron microscopy, and x-ray diffraction. Otoliths of man, rabbit, and guinea pig and rat were found to consist of calcite; those from the frog and mackerel of aragonite.

In studies on the labyrinth of pigeons, Lempert, Wever, and Lawrence [172] showed that the membranous labyrinth contains a distinctive fluid, the endolymph, and that the membranous walls constitute a barrier to the free passage of this and other fluids.

Otoliths of guinea pig and frog were studied by Matsuo and Fujiwara [185] by electron microscopy and electron microdiffraction. The following results were obtained: Very fine otolith crystals of spherical or oval form (ca. 0.4μ in length) were observed in the guinea pig and in the experimental dissolution of frog otoliths by dilute hydrocholoric acid, in the inside at first. According to the electron microdiffraction, otoliths of the guinea pig seemed to be aragonite.

Perilymph, cerebrospinal fluid and endolymph from the utricle of the guinea pig were analyzed for Na, K, Cl, and protein by Smith, Lowry, and Wu.[266] Endolymph has a K concentration 30 times that of perilymph and a Na concentration only $\frac{1}{10}$ as high as perilymph or cerebrospinal fluid. The Cl of endolymph was found to be 90% of that in perilymph and cerebrospinal fluid. Perilymph and cerebrospinal fluid were indistinguishable in K, Na, and Cl content. All three fluids are low in protein, but perilymph may contain twice as much as either of the other fluids. The tympanic boundary of the cochlear duct is at the reticular lamina covering Corti's organ. The nerve is then separated from the endolymph, which with its high K content would interfere with the conduction of the nerve fibers that cross the tunnel.

A higher content of hyaluronic acid was found in the endolymph of the vertebrate, Acanthios vulgaris, by Vilstrup and Jensen,[282] than in most other body fluids examined. The consistency of endolymph is due to a large content of this acid which is attached to proteins. The endolymph was found to contain 1.8% protein and the perilymph 1.2% protein. The albumin-globulin ratio for endolymph was albumin 0%, globulin 100%. For perilymph: albumin 21%, globulin 79%. For human serum: albumin 79%, globulin 21%. For human vitreous body: albumin 43%, globulin 57%.

The chemical composition of perilymph in cats was determined by Waltner.[289] Measurements of the ultraviolet absorption and total nitrogen were made on the perilymph and cerebrospinal fluid of 30 cats. The total N concentration was more than twice that of the cerebrospinal fluid. Contamination with blood and endolymph was ruled out. The great difference in chemical composition between perilymph and cerebrospinal fluid rules out all possibility of free flow of fluid between subarachnoidal and perilymphatic spaces. Histological studies point to the increased secretory activity in the endolymphatic spaces following drainage of the perilymph through the round window membrane. This is interpreted as indirect evidence of the role of the endolymph as source of perilymph. Localized precipitation of endolymph in the cochlear duct, in the saccule, utricle, and ampulla of the semicircular canals is considered as indirect evidence of increased production of endolymph in these anatomical structures.

An electron microscopic study by Wersall [293] of the structure of the epithelium and the nerve terminals of the crista ampullaris of the guinea pig revealed the following: two kinds of hair cells, flask-shaped short cells located on the summit of the crista, and cylindrical cells located on the periphery of the crista. About 40 sensory hairs, consisting of an axial fibril and an enveloping protoplasmic sheath, emerge from the upper surface of the sensory cells. The supranuclear part of the supporting cells contains closely packed granules. Globular protrusions emerge from the surface of the supporting cells. The flask-shaped sensory cells are supplied by coarser nerve fibers forming nerve cups about the hair cells. A large number of mitochondria are observed inside the nerve cups. There is no continuity between the cytoplasm of the nerve and of the hair cells. There is a nerve plexus, located

basally in the epithelium and consisting of fine fibers within the periphery of the crista. From this plexus emerge branches which end in club-shaped terminals connecting with the cylindrical sensory cells in the area.

A study of the ultrastructures of the labyrinthine macula was made by Coassolo.[53] The lateral semicircular canal macula, when observed with the electronic microscope, shows crystalloid formations having a high molecular mass; they derive from the protein crystalloids of the endolymph, from which they differ by a more definite degree of crystallinity.

Coassolo [54] describes his observations on the ultrastructure of the cupola of the macula utricularis. A crystalloid organic formation of high atomic weight and also a crystalloid inorganic formation of low atomic weight are identified. The latter formations are otoliths.

The central effects of cholinesters, cholinesterase, and cholinesterase inhibitors were studied by Diamant.[67] The intracarotid centripetal injection of Forssman's antigen was used in guinea pigs. In this way, the antigen reaches the cerebellum, pons, and medulla region of the injected side via the vertebral artery and causes a marked vestibular syndrome with circling. Injection of cholinesterase-inhibiting substances had the same effect. The injected side showed a lower enzyme activity in the pons medulla region. The action of anticholinesterases may be assumed to be due to their inhibiting ability. Intracarotid centripetal injections of acetycholine and mecholyl induce a vestibular syndrome, but for the induction of this syndrome the prior administration of a cholinesterase inhibitor is necessary. Damage to the vestibular nuclei produces circling towards the damaged side, whereas stimulation has the opposite effect. Injection of the cholinesterase inhibitors and cholinesters stimulates the effector cells, the result being a rotatory movement toward the opposite side. If the effector cells were destroyed by coagulation, a subsequent injection of cholinesterase inhibitor did not produce a syndrome. Destruction of the labyrinth showed that the latter was of no significance relative to induction of the syndrome. The vestibular syndrome elicited by intracarotid centripetal injection of cholinesterase inhibitors and cholinesters is of central origin. Its induction is dependent on intact vestibular nuclei in the brain stem.

In rats and dogs, the relative activity of metabolism in the labyrinthine bone was measured with P 32 and Ca 45 and compared with that in other parts of the skeleton by Steller.[267] Histologically the dog labyrinth is very similar to that of man, but that of the rat shows essential differences. The extent to which these elements are incorporated is a measure of the metabolic rate. In rats the bony labyrinth gave the same values as the parietal bone, but in dogs the labyrinth bone showed very low values. These differences are referable to the difference in histological structure.

Radioactive isotopes (iodine and phosphorus) were employed by Portmann, Portmann, and Barjon [222] to study the origin and dynamics of labyrinthine fluids. Intrathecal or intracardiac administration of radioactive isotopes to pregnant adult rats made possible the study of historadiographical variations of the labyrinths of the fetuses. Passage of the isotopes through the placenta is possible. No facts of significance on the physiology of labyrinthine fluids were revealed. Better results may be expected from this method if it proves possible in the future to use other isotopes, especially the Na of the NaCl molecule, one of the very few chemical compounds present in the labyrinthine fluid.

The problem of whether the semicircular canals play a part in directional hearing was investigated by the Meurman.[194] Bilaterally fenestrated subjects make many more errors in trying to locate the direction of a sound than do normal people. When the two ears differ in hearing, the better ear has no decisive influence on sound direction. Localization of high tones (4,000 cps) is more difficult than that of lower tones.

It is highly probable that the ampullar end-organs play a part in directional hearing.

Changes of the refractive indices of cerebrospinal and endolabyrinthine fluids in experimental and clinical hypoacusia of nervous type was studied by Hiromu.[133] Apart from deaf-mutism, no precise and definite relationship between the various types of refractive index of cerebrospinal fluid was found. Detailed animal experiments were carried out with BCG, and from the results obtained it may be concluded that there is some confirmation of Goto's theory that deafness of nervous type is often due to an allergic tuberculous mechanism.

It was possible for Gardenghi[102] to demonstrate that isoniazid, administered parenterally to pigeons, passes easily into the labyrinthine fluid, and that in these animals the drug tends to reach a higher concentration in the labyrinthine fluid than in the blood serum.

Tullio's reaction is the sequence of an inadequate stimulation of the crista ampullaris in the horizontal semicircular canal by sound vibrations, explains Huizinga.[141] After blocking the endolymph current by severing the horizontal canal in a pigeon, Tullio's reaction still occurs, if an opening is made in the small stump of the canal. Thus the stump can be compared with the normal cochlea, the opening in the stump serving as an outlet in the same way as the round window. If, after fenestration of the horizontal semicircular canal, a load is brought on the membrane of the round window, the microphonic effect of the crista increases and that of the cochlea decreases.

1229 David Whitney Building (26) (Dr. Proctor).

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CORRECTION

On page 119/**237** of the February issue of the ARCHIVES, the authors of the article entitled "Physiology of the Larynx Under Daily Stress" should be listed as Hans von Leden, M.D. (by invitation) and Paul Moore, Ph.D. (by invitation).

News and Comment

ANNOUNCEMENTS

Program for the Section on Laryngology, Otology, and Rhinology.—The 108th Annual Meeting of the American Medical Association is to be held in Atlantic City, June 8-12, 1959.

In the Section on Laryngology, Otology, and Rhinology, instead of having assigned discussers, each paper, except those of the guest of honor and the chairman, will be followed by an open discussion period; questions and comments from the floor are encouraged.

The program is as follows:

Tuesday, June 9—2 p. m.

Prof. Dr. H. H. Naumann, Würzburg, Germany
Intravital Observations of the Nasal Mucous Membrane (Film)
George M. Coates, M.D., Philadelphia
Address of Guest of Honor
Dean M. Lierle, M.D., Iowa City.
Modern Otolaryngology
F. W. Davison, M.D., Danville, Pa.
Laryngeal Obstruction in Children
Ben T. Withers, M.D., Houston, Texas
Facility in Tonsil and Adenoid Management: Conclusions from 2,400 Consecutive Cases
Joseph H. Ogura, M.D., St. Louis, Mo.
Cancer of the Pharynx, Larynx, and Upper Esophagus: Surgical Aspects
Business Meeting:
Report of Section Delegate to AMA House of Delegates
Gordon F. Harkness, M.D., Davenport, Iowa
Dean M. Lierle, M.D., Iowa City, Alternate
Report of Section Representatives to Board of Governors of American College of Surgeons
Roderick Macdonald, M.D., Rock Hill, S. C.
Edley H. Jones, M.D., Vicksburg, Miss.
John J. Conley, M.D., New York
Report of Nominating Committee
F. W. Davison, M.D., Danville, Pa.
Louis E. Silcox, M.D., Philadelphia
John D. Singleton, M.D., Dallas, Texas

Wednesday, June 10—2 p. m.

J. W. Hampsey, M.D., Pittsburgh
Current Concepts of Etiology and Management in Otolaryngic Allergy
Victor R. Alfaro, M.D., Washington, D. C.
Address of Section Chairman
Henry S. Kaplan, M.D., San Francisco
New Horizons in the Radiotherapy of Malignant Disease
John A. Kirchner, New Haven, Conn.
Facial Bone Injuries
M. R. Himalstein, M.D., Bay Pines, Fla.
Obliterative Frontal Sinus Surgery Using Gelfoam
J. M. Ravid, M.D., New York
Malignant Melanoma of the Nose and Paranasal Sinuses, and Juvenile Melanoma of the Nose
Paul H. Holinger, M.D., and Kenneth C. Johnson, M.D., Chicago
Contact Ulcer of the Larynx

Wednesday Evening, June 10—6:30 p. m.

Social Hour and Dinner—Honoring the Guest of Honor of the Section, Dr. George M. Coates

Thursday, June 11—2 p. m.

Joseph L. Goldman, M.D., and Harry Rosenwasser, M.D., New York
Current Concepts of the Management of Otitic Infections
Merle Lawrence, Ph.D., Ann Arbor, Mich.
Inner-Ear Mechanics and Deafness
Edward W. Harris, M.D., Columbus, Ohio
Symptoms Referable to the Eustachian Tube
George Kelemen, M.D., Boston
Maternal Diabetes and Congenital Deafness
Stapes Mobilization with Long-Standing Otosclerosis
Joseph Sataloff, M.D., Philadelphia
Howard P. House, M.D., Los Angeles
Polyethylene in Middle Ear Surgery

The Gould Award.—The Gould Award is presented annually for outstanding research in laryngology to encourage and reward fundamental investigations in this field.

The International Committee for the Gould Award announces the selection of the recipient for the year 1958—Prof. Fumio Nakamura, M.D., Kyoto Prefectural Medical College, Kyoto, Japan—for his electrophysiologic studies of laryngeal function.

The International Committee for the Gould Award for 1959 will consist of Sir Victor Negus, 149 Harley St., London, W. 1; Professor Cotoji Satta, No. 111 Morikawa-cho, Bunkyo-ku, Tokyo; and Hans von Leden, M.D., Medical Director, The William and Harriet Gould Foundation, 30 N. Michigan Ave., Chicago 2.

The amount of the Award has been increased to $300.

GENERAL NEWS

American Medical Writers' Association Elects 1959 Officers and Directors.—At the 15th annual meeting of the American Medical Writers' Association, held at the Hotel Morrison, Chicago, Sept. 26-27, 1958, the following officers and directors were elected to serve for 1959:

President-Elect: Austin Smith, M.D., Chicago
First Vice-President: Karl A. Menninger, M.D., Topeka, Kan.
Second Vice-President: Theodore R. Van Dellen, M.D., Chicago
Editor: Raymond V. Pogge, M.D., Cincinnati (Reelected)
Accounting Officer: Alphonse McMahon, M.D., St. Louis (Reelected)
Secretary-Treasurer: Harold Swanberg, M.D., Quincy, Ill. (Reelected)
DIRECTORS:
John A. Wagner, M.D., Baltimore (Two-year term)
Col. John B. Coates, M.D., Washington, D. C. (Reelected two-year term)
Norris J. Heckel, M.D., Chicago (Reelected two-year term)
Don C. Lyons, D.D.S., Jackson, Mich. (Reelected one-year term)
W. D. Snively, Jr., M.D., Evansville, Ind. (Reelected two-year term)
Julian A. Sterling, M.D., Philadelphia (Reelected two-year term)
George G. Stilwell, M.D., Rochester, Minn. (Reelected two-year term)

Morris Fishbein, M.D., Chicago, formerly Editor of *The Journal of the American Medical Association*, is the 1959 President, having been elected last year. C. E. Lyght, M.D., Rahway, N. J., is the immediate Past President. Lee D. van Antwerp, M.D., Chicago, was reelected Chairman of the Board of Trustees, and Frank B. Ramsey, M.D., Indianapolis, was reelected Chairman of the Advisory Committee. Charles E. Lyght, M.D., Rahway, N. J., retiring President, and Lee D. van Antwerp, M.D., Chicago, Trustee, were elected members of the Executive Committee.

Books

BOOK REVIEWS

Sprachhörprüfmethoden: Grundlagen, Würdigung und Anwendung bei Begutachtund und Hörgeräteanpassung. By Prof. Dr. Med. Dipl.-Ing. Kurt Schubert. Price, $16.45. Pp. 361, with 187 illustrations. Georg Thieme Verlag ,Herdweg 63, (14a) Stuttgart. (American agent—Grune & Stratton, Inc., 381 4th Ave., New York 16), 1958.

The author, an acoustical engineer as well as an otolaryngologist, gives a thorough review of all aspects of speech audiometry. He uses for testing an audiometer devised by himself and electromagnetically recorded groups of meaningful and meaningless words specially selected by him which are, as to the percentual frequency and the distribution of the contained sounds, representative of the average German language. The technical requirements for satisfactory speech audiometers and testing rooms are discussed in detail. The influence of a frequency shift in the test words above and below the usual level is investigated, the methods of speech audiometry reviewed, and the results achieved with the author's groups of test words reported. The factors influencing the audibility of sounds are analyzed and the advantages and disadvantages of testing with numbers pointed out. The author then describes his method of determination of the amount of hearing loss for speech and the resulting degree of disability. The physical principles governing the relationship between direct and reflected sound in closed rooms and the influence of these factors on the subjective perception of sound are discussed. The author then describes his testing method, in which the speech tests are given not only with ordinary sound but also with mixtures of pure and reflected sound ("Hall-Audiometrie"). The clinical applications of this method are demonstrated. This is followed by a detailed description of the methods of hearing aid selection. A discussion of the speech-audiometric tests for detection of malingering concludes the book.

Limitations of space do not permit a detailed discussion of the many interesting results of the author's investigations. Although the book deals with German speech audiometry, it is also of great interest to the English-speaking audiologist.

La scoperta degli ossicini auricolari. (The Discovery of the Auditory Ossicles). By Alessandro Fioretti and Giuseppe Concato. Price, not given. Pp. 114, with 11 illustrations. Collana Monografica di Minerva Otolaryngologica, Torino, Italy, 1958.

The authors examined a considerable part of the original sources to build up their survey on the first events around the discovery of the auditory ossicles and the numerous controversies regarding priority and other issues. Facsimiles of portraits and of anatomical illustrations enliven the text, and the summary is given in an English translation. It was no mean task to disentangle the labyrinth of data leading to a final establishment of knowledge around this section of aural anatomy. This is a very timely, highly readable, and instructive monograph.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.
Meeting: 24th Annual Congress, Palmer House, Chicago, Sept. 14-17, 1959.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Meeting: July 15, 1959, New York.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.

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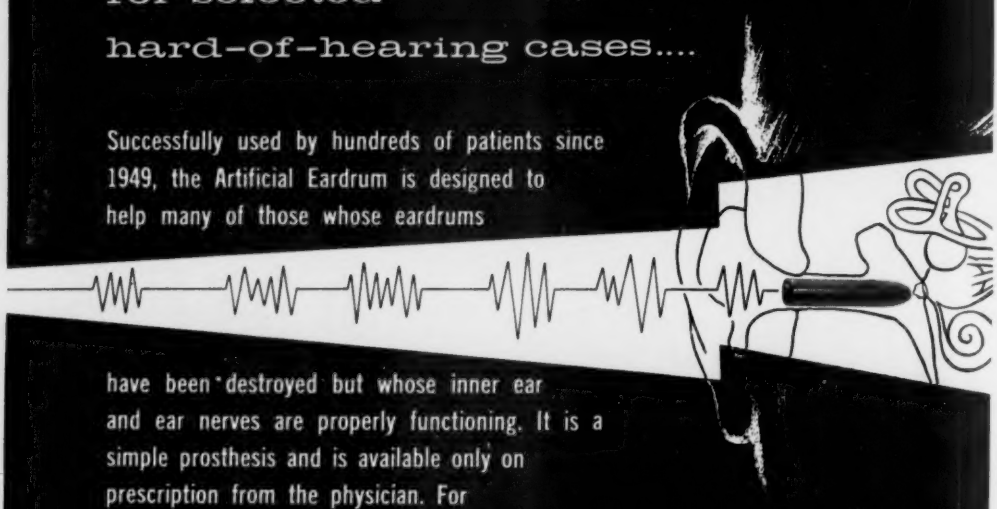

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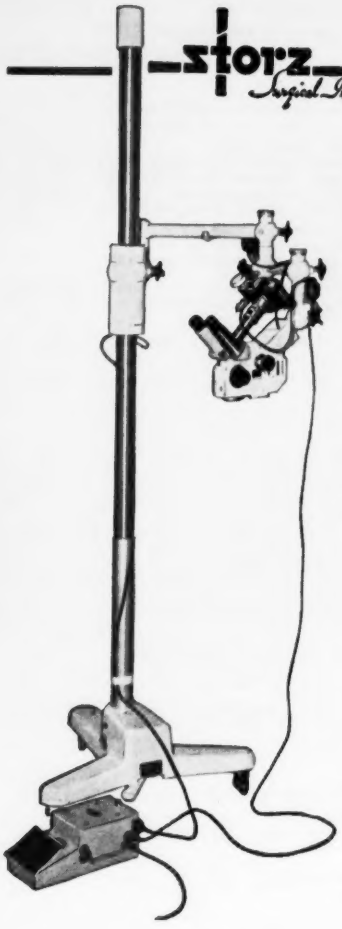

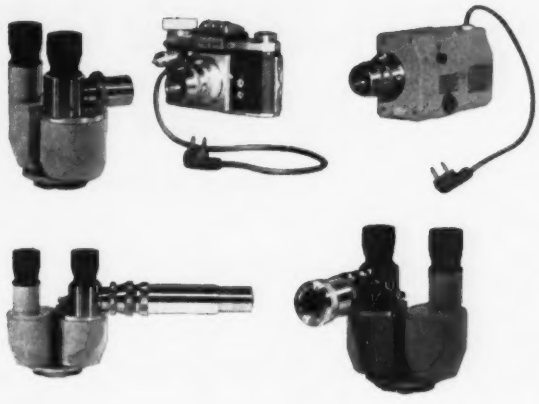

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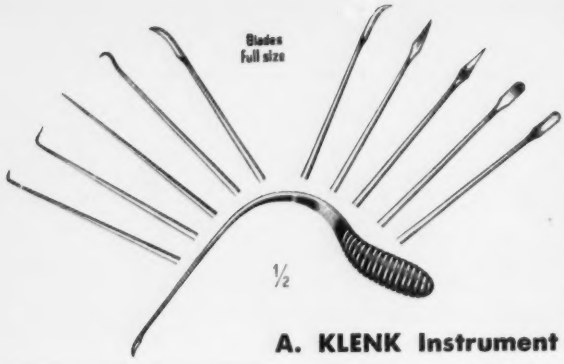

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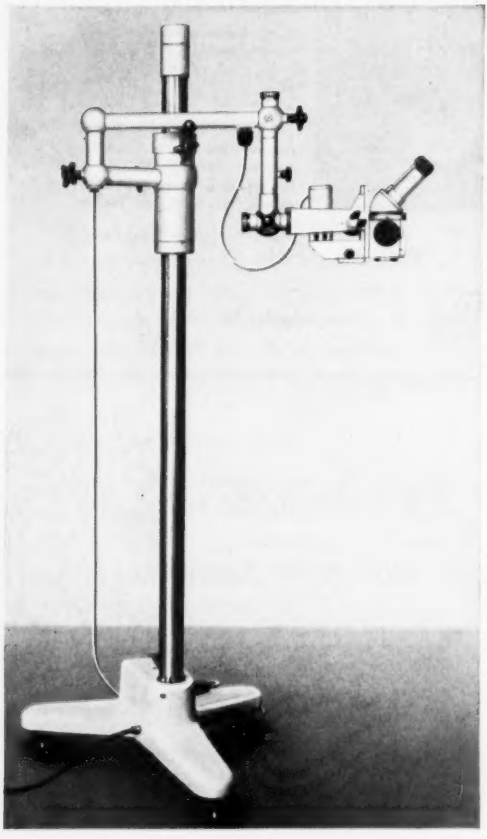

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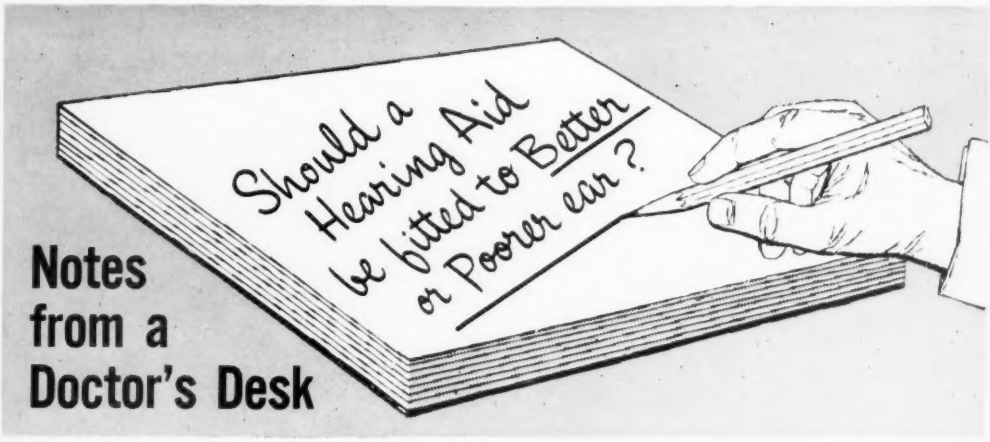

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